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Teque auscultantem palpantem et percutientem
Pectora, sic morbi ducere signa vident.

E Carmine Roberti Bridges
De nosocomio Sti Bartolomaei Londinensi.

OXFORD MEDICAL PUBLICATIONS

AUSCULTATION AND PERCUSSION

TOGETHER WITH

THE OTHER METHODS OF PHYSICAL EXAMINATION OF THE CHEST

BY

SAMUEL GEE, M.D.

Fellow of the Royal College of Physicians, Honorary
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Bartholomew's Hospital

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TO

THE MEMORY

OF

LAENNEC.

E TENEBRIS TANTIS TAM CLARVM EXTOLLERE LVMEN
QVI PRIMVS POTVISTI INLVSTRANS PECTORIS ANTRVM
TE SEQVOR O GALLAE GENTIS DECVS INQVE TVIS NVNC
FIXA PEDVM PONO PRESSIS VESTIGIA SIGNIS
NON ITA CERTANDI CVPIDVS QVAM PROPTER AMOREM
QVOD TE IMITARI AVEO.

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PREFACE

TO FIFTH EDITION.

THIS book is a record of the methods, facts and doctrines which the author, for many years, has found useful in the practice of his profession. No doubt the time has come when skiagraphy by X-rays should be reckoned among the methods of physical examination of the chest, yet inasmuch as few medical men possess the necessary apparatus, the topic has been excluded from the present book.* Great pains have been taken to use technical words in their proper and original meaning. The sense of words clearly defined has not been perverted, nor have new words been introduced to denote signs already well denominated. Much of the difficulty of teaching auscultation and percussion to students is due to neglect of these plain rules.

---

* A complete treatise on this subject is about to be published by Dr. Hugh Walsham and Dr. Geo. Harrison Orton: "The X-rays in the diagnosis of diseases of the chest."

The pleasing duty remains for the author to acknowledge the help which he has received in the preparation of this edition from Dr. T. J. Horder.

August, 1906.

TABLE OF CONTENTS.

Page
INTRODUCTORY CHAPTER 1

PART THE FIRST.

Chap. I. Method of Examination 3
„ II. Inspection 4
Sect. I. Shape of Chest at Rest . . 4
Art. I. Shape in Health . . . 6
¶ I. Typical Shape 6
II. Subtypical Shapes . . 10
i. Alar chest 11
ii. Flat chest . . . 12
iii. Transverse Constriction . 14
iv. Pigeon breast . . . 16
v. Rickety chest . . . 19
Art. II. Shape in disease . . . 20
¶ I. Bilateral changes . . . 21
i. Enlargement . . . 21
ii. Diminution . . . 25

Chapter II.—*continued.*

Page
¶ II. Unilateral changes . . 26
i. Enlargement . . . 26
ii. Diminution . . . 29
¶ III. Local changes. . . . 31
i. Bulging . . . 31
ii. Shrinking . . . 32
Cup-shaped depression 32
Sect. II. Movements of Chest . . . 34
Art. I. Movements of Respiration . . 34
¶ I. In Health 34
II. In Disease 36
i. Inspiratory Dyspnœa . . . 36
ii. Expiratory Dyspnœa . . 37
iii. Non-expansive Inspiration . 37
iv. Respiration wholly Thoracic . 38
v. Respiration wholly Abdominal 38
Art. II. Movements of the Heart . . 38
¶ I. In Health 39
i. The Impulse 39
ii. Recession 40
¶ II. In Disease 40
i. The Impulses 40
α. Of the apex-beat . . 41
β. Of the conus arteriosus . 42
γ. Of the right auricle . 42
ii. Recession 43
Art. III. Movements wholly unnatural . 44

Page

Chap. III. Palpation 45
  Sect. I. Lungs and Pleuræ 45
    Art. I. Vocal Thrill 45
      ¶ I. In Health 45
      II. In Disease 46
    Art. II. Pleural Friction 47
    III. Fluctuation 47
  Sect. II. Heart and Pericardium . . . 48
    Art. I. Impulses 48
      ¶ I. Systolic 48
      II. Diastolic 50
      III. Præsystolic 50
    Art. II. Valvular Thrills 51
    III. Pericardial Friction . . . 53
    IV. Fluctuation 54
  Sect. III. Large Vessels 54
Chap. IV. Percussion 55
  Sect. I. Introductory 55
    Art. I. Historical 55
    II. Method 58
      i. Immediate Percussion . . 58
      ii. Mediate Percussion . . 59
    Art. III. Theory 60
      ¶ I. Percussion Sounds 61
        Class I. Percussion Tones . . 61
          I. Loudness 61
          II. Duration 62

Chapter IV.—*continued.* Page
- III. Pitch 62
  - Tympanitic, pulmonal, tracheal and osteal tones 62
- IV. Tone 64
  - Clearness and Dulness . 64
- Class II. Percussion Noises. . 65
  - III. By-sounds . . . 65
    - I. Metallic Ring. . 65
    - II. Cracked-Pot Sound 65
- ¶ II. Physical Conditions of—
  - I. Percussion Tones . . . 66
  - II. Percussion Noises . . . 72
  - III. By-sounds. 72
- ¶ III. Percussion Resistence . . . 74
  - Percussion Thrill 75
  - Superficial and Deep Percussion . 75
- Sect. II. Percussion of Chest in Health . . 76
  - Art. I. Pulmonary Region . . . 77
    - ¶ I. Resonance and Resistence . . 77
    - II. Extent. 78
  - Art. II. Cardiac Region 79
  - III. Mediastinal Region . . . 81
- Sect. III. Percussion of Chest in Disease. . 81
  - Art. I. Pulmonary Region . . . 81
    - ¶ I. Percussion Sound 82
      - Class I. Increased Resonances . . 82
      - II. Diminished Resonances . . 83

Chapter IV.—*continued.* Page

Class III. By-Sounds 85
¶ II. Percussion Resistence . . . 85
III. Extent 86
Art. II. Cardiac Region 86
i. Area diminished 86
ii. Area increased 86
Art. III. Mediastina 87
Chap. V. Auscultation 88
Sect. I. Introductory 88
Art. I. Historical 88
II. Methods 91
III. Murmurs in general . . . 94
Sect. II. Lungs and Pleuræ 95
Art. I. Vocal Resonance 96
¶ I. Theory 96
i. Muffled vocal resonance . . 97
ii. Bronchophony 97
Pectoriloquy 99
Ægophony 100
II. Physical Conditions . . . 101
III. Vocal Resonance in Health . . 108
IV. Vocal Resonance in Disease . . 109
Resonance of Cough and Cry . 110
Art. II. Respiratory Sounds 111
¶ I. Theory 111
i. Vesicular breathing . . . 111

Chapter V.—*continued.* Page

ii. Bronchial breathing . . 112
Cavernous and tubular . . 114
Souffle voilé . . . 115
¶ II. Physical Conditions . . . 115
III. Respiratory Sounds in Health . 118
IV. Respiratory Sounds in Disease . 119
V. Rales 121
i. Crepitant 121
ii. Mucous 123
iii. Sonorosibilant 124
iv. Doubtful 124
Art. III. Pleural Sounds 125
¶ I. Friction Sounds 125
II. Amphoric Sounds 128
i. Amphoric hum . . . 128
ii. Metallic tinkle 129
iii. Bell sound 129
iv. Splashing sound . . . 130
v. Physical conditions . . 130
Appendix to Sect. II. 132
i. Peritonæal friction 132
ii. Shoulder-joint friction 132
iii. Shoulder-blade friction . . . 133
iv. Muscular rumble 133
Sect. III. Heart and Pericardium . . . 133
Art. I. Sounds in Health 135
Loudness and Accentuation . 136

Chapter V.—*continued.* Page

Art. II. Sounds in Disease 137
¶ I. Murmurs 138
Class I. Cardiac Murmurs . . 139
i. Place 141
ii. Time 144
iii. Meaning 146
iv. Loudness 147
v. Reduplication 149
i. Simple 149
ii. Reduplication Murmurs 151
Class II. Vascular Murmurs . . 151
¶ II. Pericardial Sounds . . . 152
I. Friction 152
II. Waterwheel sounds . 155
Appendix to Sect. III. 156
¶ I. Pulsatile Pulmonary Sounds . . 156
II. Pulsatile Friction Sounds . . . 158
III. Metallic Jingle 158
Chap. VI. Appendix to Part I. 160
Sect. I. Auscultation of Arteries . . 160
I. Conducted sounds 160
II. Sounds produced in situ . . . 160
¶ I. Spontaneous 160
i. Aneurysmal 160
ii. Subclavian 161
¶ II. Factitious 161
i. Systolic 161
ii. Diastolic 162

Chapter VI.—*continued.* Page
Sect. II. Inspection of Veins of Neck . . 163
¶ I. Fulness of Veins 163
II. Venous Pulsation 164
Sect. III. Auscultation of Veins of Neck . 170
¶ I. Continuous Venous Hum . . 170
II. Intermitting Murmurs . . . 171
Sect. IV. Epigastric Pulsation 171
V. Position of Diaphragm . . . 173
¶ I. In Health 173
II. In Disease 174
Sect. VI. Position of Mediastinum . . . 175
VII. Other Vascular Murmurs . . 177
VIII. Puncture of the Chest . . . 179
IX. Conduction of Respiratory Sounds 182
Auscultation of Œsophagus . . 183

PART THE SECOND.

Chapter I. Pulmonary Catarrh, or Bronchitis .
II. Pulmonary Œdema . . . . .
III. Pulmonary Congestion . . .
IV. Pulmonary Hæmorrhage . . .
V. Pulmonary Emphysema . . .
VI. Pulmonary Atrophy . . .
VII. Asthma . . . . .
VIII. Pulmonary Collapse . . .
IX. Plugging of Trachea or Bronchus
X. Pleurisy . . . . .

Chapter Page
XI. Pneumothorax 206
I. Closed Hydropneumothorax . 206
II. Fistulous Empyema. . . 209
III. Loculated Pneumothorax . . 210
IV. Pure Pneumothorax . . . 210
XII. Hydrothorax 213
XIII. Pleurisy with Effusion . . . 214
XIV. Empyema 227
XV. Adherent Pleura. 232
XVI. Pneumonia 234
I. Lobar Pneumonia . . . 234
II. Lobular Pneumonia . . . 238
XVII. Destructive Pneumonia and Pulmonary Gangrene 239
XVIII. Embolic Pneumonia or Pyæmic Infarctus 241
XIX. Pulmonary Tuberculosis . . . 242
XX. Pulmonary Phthisis 244
XXI. Pulmonary Cancer 249
XXII. Pulmonary Hydatids 252
XXIII. Pulmonary Actinomycosis . . . 254
XXIV. Dilatation of the Bronchi . . . 255
XXV. Pulmonary Cirrhosis 256
XXVI. Hypertrophy of the Heart . . . 258
XXVII. Dilatation of the Heart . . . 260
XXVIII. Pericarditis 263
XXIX. Pericardial Effusion 265
XXX. Pneumopericardium 271

Chapter Page
XXXI. Adherent Pericardium 272
XXXII. Mitral Regurgitation 273
XXXIII. Mitral Obstruction 276
XXXIV. Aortic Regurgitation 280
XXXV. Aortic Obstruction 284
XXXVI. Tricuspid Regurgitation 285
XXXVII. Tricuspid Obstruction 286
XXXVIII. Pulmonary Regurgitation . . . 287
XXXIX. Pulmonary Obstruction 289
XL. Murmurs of Uncertain Nature . . 290
XLI. Malformations of the Heart . . 293
XLII. Mediastinal Tumours 295
XLIII. Aneurysm of the Thoracic Aorta . 297
XLIV. Other Intrathoracic Aneurysms . . 304
I. Innominate Artery . . . 304
II. Pulmonary Artery . . . 305

INDEX 307

PHYSICAL EXAMINATION OF THE CHEST.

INTRODUCTORY.

PHYSICAL EXAMINATION AND PHYSICAL SIGNS.

PHYSICAL Examination relates to those phænomena which a person examining can discover for himself by means of his own senses, and without any reference to the sensations of the patient. Most parts of the body may undergo a physical examination; the immediate results thereby obtained are called Physical Signs.[1]

---

[1] "When a man hath so often observed like antecedents to be followed by like consequents, that whensoever he seeth the antecedent, he looketh again for the consequent; or when he seeth the consequent, maketh account there hath been the like antecedent; then he calleth both the antecedent and the consequent, *signs* one of another." Hobbes: Human Nature, Chap. IV. par. 9. Cf. Computation or Logic, Chap. II. par. 2.

The present book is devoted to an exposition of the methods and results of physical examination of the organs contained in the thorax: namely, of the lungs and pleuræ, the heart and pericardium, and the mediastinum, including the large bloodvessels.

The First Part treats of the physical signs considered in the abstract; the pure science of the physical signs.

The Second Part treats of the physical signs considered in their subservience to the discovery of disease; the applied science of the physical signs.

PART THE FIRST.

CHAPTER I.

METHOD OF EXAMINATION.

SUPPOSE a patient with the chest exposed, ready to undergo a physical examination : the physician first of all carefully surveys the chest with his eye, this is Inspection : next, with his hand, this is Palpation : he next strikes the chest, Percussion : and lastly he puts his ear upon the chest, Auscultation.

Whenever convenient, the patient should remove all clothes from the upper part of the body down to the waist, and stand opposite to the physician. Needful deviations from this rule will be suggested by the good sense of the examiner at the proper time.

CHAPTER II.

INSPECTION.

INSPECTION discovers the shape of the chest. First; the shape such as it is when the thorax is at rest; that is to say, at the end of an ordinary expiration and during the diastole of the heart. Secondly; the ceaseless temporary changes in shape which the chest undergoes during life, in consequence of the respiratory and circulatory movements.

SECTION I.

SHAPE OF THE CHEST AT REST.

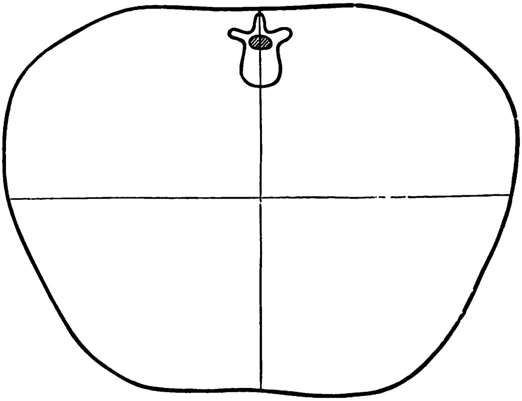
A transverse section of the chest upon a horizontal plane approaches to the figure of an ellipse; between the long and short axes of which (that is to say, between the breadth and depth of the chest) there is a certain proportion. Knowledge of this proportion is the key to knowledge of the shape of the chest in health, and the unilateral and bilateral changes which that shape undergoes in disease. Changes in the length or height of the chest from above downwards, changes in the direction of the ribs, in the width of the intercostal spaces, in the size of the costal angle, in the arching of the spine and sternum, in the height of the shoulders, and in the projection of the shoulder-blades ; all these have a definite relation to changes in the shape of the horizontal ellipse.

When the axes of the ellipse are nearly equal in length, and the horizontal plane is nearly circular in outline, the chest is short from above downwards (the floating ribs being excluded from consideration), the ribs approach the horizontal, the intercostal spaces in front are narrow, the costal angle is obtuse or open, the sternum is arched, the shoulders are high, and the scapulæ lie flat on the ribs. On the other hand, in proportion as the transverse axis of the ellipse exceeds in length the anteroposterior, the chest becomes long, the ribs slope downwards, the intercostal spaces in front are wide, the costal angle is acute, the sternum is straight, the shoulders are low, and the corners of the shoulder-blades project from the ribs. The former is the chest of inspiration or expansion, the latter is the chest of expiration or contraction.

ARTICLE I.—SHAPE IN HEALTH.

We may conceive an idea or a type of what the perfect human chest should be. But the ideal shape is seldom realised ; deviations from the type are present in nearly all persons, who nevertheless may be in soundest health. The more important of these deviations will be described under the title of subtypical.

Fig. 1.



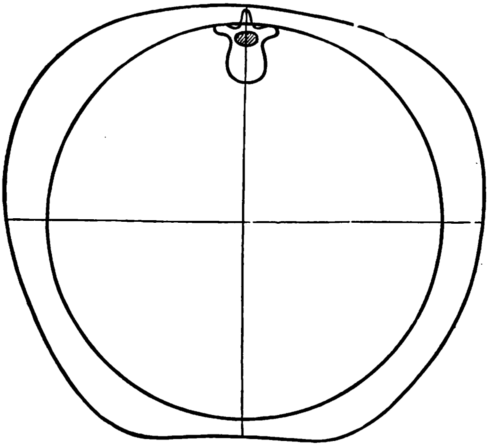
Circumference = 89 centimeters.

Transverse section of healthy adult chest upon level of sternoxiphoid articulation.

¶ I.—THE TYPICAL SHAPE.

In new-born children the axes of the ellipse are almost equal ; the thorax is nearly as deep as it is broad, and is not far from circular. As growth proceeds the breadth of the chest increases more quickly than the depth ; so that, by the time the child has cut his milk teeth, a strongly elliptical shape is established. The disproportion between the axes becomes greater and greater (but with diminishing rate of yearly increase) until the body is fully developed. After maturity no further changes occur,

Fig. 2



Circumference = 40·5 centimeters.

Transverse section of chest of an infant aged 9 months.
(A circle drawn within the tracing for the sake of comparison.)

until those morbid conditions, which are the almost necessary companions of old age, begin, in most persons, to alter the typical shape, and to make the chest acquire, in the second childhood, much the same figure that it had in the first.

The Cyrtometer is an instrument by means of which the shape of the chest may be exactly ascertained and registered. Originated, both in notion and in name, by Félix Andry,[1] it was by Woillez[2] that the cyrtometer was first made really useful in physical diagnosis. I have introduced a cheap and perfect cyrtometer which consists of two pieces of composition gas-pipe, drawn out to a diameter of the eighth of an inch, and united by a piece of caoutchouc tubing. The instrument, after having been accurately applied to a given circumference of the chest, is removed, and will·then afford a tracing of that circumference. The composition alloy is slightly, though but slightly, resilient: wherefore it is well to determine the antero-posterior diameter of the chest by means of calipers, and so to correct the cyrtometer before tracing the outline. The arms must hang by the sides of the chest while the cyrtometer is adapted thereto : for when the arms are raised or held forward the shape of the chest is changed. It is needless to add that the cyrtometer is the best means of measuring the chest.

---

[1] Manuel de diagnostic des maladies du cœur: Paris, 1843. A book which I have not been able to procure.

[2] Recherches cliniques sur l'emploi d'un nouveau procédé de mensuration dans la pleurésie. Paris, 1857.

In order to illustrate the statements just made respecting the different shapes of the thorax at different ages, I will give some actual measurements, taken upon the level of the sternoxiphoid joint, and so calculated that the circumference always = 100.

Age.	Actual circumference.	Ratio of diameters to circumference.	
		Antero-posterior.	Transverse.
3 months...	$14\frac{3}{4}$ inches (37·5c.) ...	26	29
2 years......	18 ,, (45·75c.)...	26	32
34 years......	$29\frac{3}{5}$,, (75c.)	26	35
48 years......	35 ,, (89c.)	27	31

The chests measured were all perfectly healthy, saving the last, which was emphysematous.

The semicircumference of the right half of the chest is usually a trifle greater than that of the left ; the maximum of difference, on the nipple level, being an inch and a quarter. The nipples are seated on the fourth interspaces. The manubrium joins the body of the sternum at an angle, level with the second rib ; the angulus Ludovici,[1] which affords an easy and trustworthy guide in counting the ribs.

It is convenient to regard the chest as mapped out by certain vertical lines, whereby we can indicate the exact longitudinal situation of any physical sign. The following vertical lines will be found sufficient : the midsternal, right or left side-sternal, parasternal (i.e. midway between the side-sternal and nipple lines), nipple, mid-axillary, scapular (i.e. the angle), and the vertebral groove. The horizontal level or latitude is indicated by reference to the clavicles, ribs, intercostal spaces, nipples, and sternoxiphoid articulation.

¶ II.—SUBTYPICAL SHAPES.

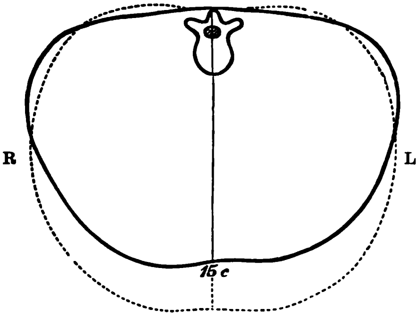
There are certain deviations from the typical shape of the chest which are present in a large number of persons free from any disease of the thoracic organs. And, partly for this very reason, but chiefly because these deformities indicate pulmonary disease in the past, or a tendency to it in the future, they are worthy of attention. They are of five kinds, to wit, the alar or pterygoid, the flat, the transversely constricted, the pigeon, and the rickety chests.

---

[1] The origin of this term is uncertain : it is commonly supposed to refer to P. C. A. Louis, but I have not met with the least allusion to the sternal-ridge in any of his writings.

i. THE ALAR CHEST.—It has been known from of old that many persons predisposed to phthisis manifest their predisposition by an unnaturally small chest. Projection of the angles of the scapulæ, so as to look like wings, is one sign of the small capacity of these chests,

Fig. 3.



Circumference = 59·5 centimeters.

ALAR THORAX.

Tracing taken from a child. The dotted line indicates the shape of the chest of a healthy child of the same size. The circumferential measurement refers to the alar thorax.

which are therefore called alar or pterygoid.[1] The thorax of phthinodes (persons predisposed to phthisis) is, as Galen[2] says, narrow and shallow; the anteroposterior diameter is especially small. This diminution in capacity is brought about by drooping, or undue obliquity of the ribs, hence the shoulders fall, and the length of the thorax from above downwards is increased; it is the falling of the shoulders which causes the alar appearance. The pterygoid chest is often accompanied with a prominent throat, as Aretæus[3] says, due to a long neck, and the head being carried unduly forwards.

ii. THE FLAT CHEST.—The diminution in capacity presented by the alar chest does not necessitate any great change in the outline which a horizontal plane of the thorax naturally presents. But sometimes not only the size of the sectional area but its shape also is changed, by the cartilages of the true ribs losing their curve and becoming straight, in which case the chest looks quite flat in front instead of being rounded, the horizontal ellipse is flattened from before backwards, nay sometimes the sternum is depressed below the level of the cartilages so that a section would be somewhat kidney-shaped, the cartilages being curved in the wrong direction. In other respects the flat chest mostly presents the characters of the alar thorax ; but this is not always the case, inasmuch as the alar appearance is due to increased obliquity of the ribs and falling of the shoulders, conditions which are not always present even in a well-marked flat chest, the diminution of capacity being otherwise brought about.

---

[1] Hippocrates: Epidemics, bk. vi. sect. iii. par. 10.

[2] Comment. in Hippocr. Epidem. i. pag. 62 (edit Kühn).

[3] Caus. et sign. morb. chron. bk. i. ch. 8.

Both the phthinoid chests (alar and flat) are often modified in shape by the presence of the transverse constriction to be hereafter described. And both are, as Van Swieten[1] says, essentially the same as the actually phthisical chest, but deformed to a less degree ; moreover the loss of fat and muscle which occurs in phthisis makes all the characters described more obvious.

The phthinoid chests are Natural deformities, the tendency to which is born with the individual and inseparable from him. I now come to the Accidental deformities of the chest, those which have been produced by actual disease subsequent to birth.[2]

---

[1] Comment. in Aphor. 1198.

[2] " Naturalem formationem eam appello quæ sit cum

iii. Transverse Constriction of the Chest.—A deformity from which few persons are wholly free. It consists in a depression, more or less deep, of the chest walls in front, which passes outwards and slightly downwards, on both sides, level with the xiphoid cartilage, and ceases gradually towards the mid-axillary line.

Produced during childhood, the groove simply persists in after-years. Its immediate cause is an impediment to the inspiration of air sufficient to fill the whole of the lungs. In the chapter on inspiratory dyspnœa it is explained that the upper part of the chest can be expanded and kept expanded much more powerfully than the lower part can be. Hence when the diaphragm descends and the thorax, for any reason, cannot hold out against the vacuum which would otherwise be thus created, the lower part of the chest is the first to yield to the atmospheric pressure upon it from without. This comparatively feeble expansion of the lower thorax becomes manifest when, during inspiration, an insufficient supply of air enters

---

pectore constricto, longo collo, et humeris alatis ; accidentalem vero quæ sit cum curvitate seu distortione pectoris." Morton : Phthisiologia, bk. ii. chap. i. Lond. 1689.

the lungs in consequence of obstruction in the respiratory passages. What air can enter goes to the part of the chest most powerfully kept expanded, so that the feeble lower thorax gets little or none, and succumbs to the external pressure. Catarrh is that obstruction in most cases. Nor need the catarrh be at all severe, or the impediment at all great, inasmuch as a necessary concurrent cause of the groove is found in the yielding character of the ribs during infancy and early childhood, and especially of ribs rendered (as they so often are) præternaturally yielding by rickets.

When the impediment is severe and protracted, the depression, although proportionally great, ceases to exist alone ; other deformities are produced, a pigeon breast, or a cup-like hollow.

But when the deformity stops short of a pigeon breast, that is, when the depression is not so great as to involve the whole of the front base of the thorax from the xiphoid level downwards, it is the abdominal viscera which determine the position of the sulcus by maintaining the expansion of the base of the chest. The depression occurs as low down as possible, namely, immediately above the upper surface of the abdominal viscera, or what comes to the same thing, the sulcus corresponds to the vault of the diaphragm. This fact led Edwin Harrison[1] to propose the sulcus as an easy means of determining the upper margin of the liver. But be it remembered that the groove indicates what was the upper margin of the liver in early life ; and although the relationship of the parts concerned is scarcely altered in adult age, excepting from disease, yet any change in the position of the liver, after the furrow has been formed, does not change the position of the furrow itself.

iv. THE PIGEON BREAST.—The essential character of the pigeon breast is a straightening of the true ribs in front of their angles. Connected with this deviation from the natural shape are two other changes, namely, first, that the sternum is thrown forwards, and next, that the greatest transverse diameter of the chest recedes towards the costal angles, that is, that the horizontal section tends to pass from the ellipse into the triangle.

The cause of pigeon breast is a long existing or frequently recurring impediment to free inspiration while the ribs are yielding, that is, during childhood; and especially when they are præternaturally yielding, that is when rickety. Chronic pulmonary catarrh (including hooping cough) and chronic enlargement of the tonsils are common causes of pigeon breast: by watching the chest of a child during the long-drawn inspiration of hooping cough, the deformity may be seen in process of formation.

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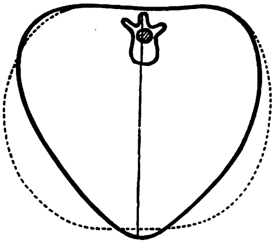
[1] London Med. Gaz. for 1836, vol. xix. pp. 369 and 776.

Fig. 4.



Circumference = 57·5 centimeters.

PIGEON BREAST.

Tracing taken from a child of seven years. Dotted line indicates natural shape at same age.

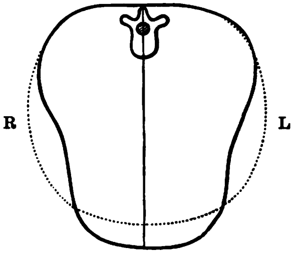
Obstructed indraught of air renders forced inspiration needful: forced inspiration over-expands the upper thorax: over-expansion of the thorax is marked by protrusion of the sternum, a necessary character of pigeon breast. The production of the peculiar note of that deformity, the straightened ribs, is not so easily understood. We have to explain why forced inspiration should produce, sometimes a simply dilated chest with arched ribs, and other times the more complex deformity with straightened ribs, called pigeon breast. Something more than mere softness of ribs must be concerned: for either shape may be produced in the youngest children, under conditions which seem to be the same in both cases, although the results differ.

But obstruction to inspiration was pronounced to be the cause of the transversely constricted chest. And so a pigeon breast is mostly accompanied by a well-marked transverse sulcus: as a part of which transverse constriction, the xiphoid cartilage becomes bent back so as to form a more or less sharp angle at the bottom of the sternum; a condition which increases the similarity to a pigeon's breast. The transverse furrow, I say, is well marked; in fact, very often there is a depression of the whole anterior half of the thorax below the xiphoid level; in which case, the straightening of the false ribs, in front of the angles, is carried to an extreme degree.

The costal cartilages of a pigeon breast are often more prominent on the right side than on the left: a condition sometimes connected with more or less scoliosis in the dorsal vertebræ; sometimes dependent upon the fact that where on the right side is lung, free to dilate, there, on the left side, lies the heart.

v. THE RICKETY CHEST.—Rickets, on account of the part which it takes in the generation of the transversely constricted and of the pigeon chest, has already been alluded to more than once: it remains to show how rickets alone may produce deformity of the chest.

Fig. 5.



Circumference = 42·75 centimeters.

RICKETY CHEST.

Dotted line indicates shape of chest in an infant of about the same age.

Rickets is a disease of infancy, and infants cannot but have a respiration chiefly abdominal, because of the circular shape of their thorax, which does not admit of further horizontal expansion. When the diaphragm descends and rarefies the air contained in the lungs, the rickety ribs, not being able to hold out until the chest is completely distended by fresh air passing in through the glottis, yield in their softest parts to the atmospheric pressure from without, and are bent inwards. Inasmuch as the softest parts of the ribs are at and near their costochondral articulations, a shallow longitudinal groove is formed on each side of the chest, more or less parallel with the sternum and ending just above the costal margin: a groove which may be formed without the least direct impediment to the entry of air through the air-passages.

Deformities of the chest which are purely rickety tend to disappear to a remarkable degree as the health improves: deformities of more complex nature are more permanent.

ARTICLE II.—SHAPE IN DISEASE.

Having described the deviations from the natural shape of the chest which are compatible with a healthy state of its contents, I now come to those changes in shape which indicate disease of the thoracic viscera. Changes of this latter kind may be reduced into three classes, namely, bilateral, unilateral, and local. The former two classes of change indicate disease of the lungs or pleuræ: the last class of change may be caused by disease of lungs, heart, serous membranes, or mediastina.

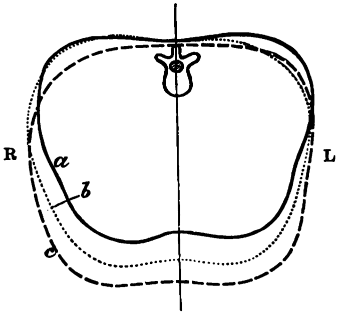
¶ I.—BILATERAL CHANGES IN SHAPE:

are of two kinds, enlargement and diminution.

i. BILATERAL ENLARGEMENT.—By the deepest inspiration (in other words, by the greatest elevation and rotation of the ribs) no considerable change can be produced in the proportion between the length of the two axes of the horizontal ellipse. In order to render further enlargement of the thorax possible the ribs must change their shape; they become more curved: the axes of the ellipse tend to become equal; the ellipse tends to pass into the circle; changes which are explained by the fact that of all figures possessing a periphery of fixed and certain length, the circular is that which includes the greatest area; depart from the circle in any way, and the area becomes less. In order to produce bilateral enlargement of the chest, forced inspirations must be incessantly repeated for a length of time sufficient to expand the thorax to a degree beyond that which is possible by a single forced inspiration of a healthy chest. The experiment indicated by the annexed diagram (fig. 6) illustrates this point.

Enlargement of the chest signifies enlargement of its contents; and the only disease which fulfils this condition is emphysema of the lungs. In a well-marked case of emphysema, then, the thorax is in a state of distension beyond what could have been produced during health by the deepest inspiration. The chest is almost cylindrical or semi-globular, arched before and

Fig. 6.



Horizontal section of chest of a child two years old.

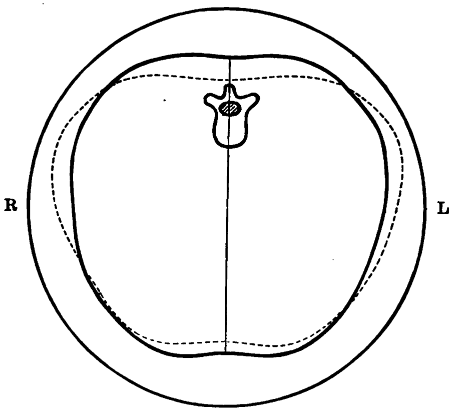
a = chest at rest.
b = chest after fullest expansion possible of lungs.
c = chest after forcible injection of air into both pleural cavities.

Anteroposterior diameters:
$a = 10{\cdot}2$c. $b = 11{\cdot}7$c. $c = 12{\cdot}2$c.

Circumferences:
$a = 47{\cdot}5$c. $b = 48$c. $c = 48{\cdot}5$c.

behind. The arching is usually most marked in the sternum, and is simply the result of the fact that the sternum is less able to move forwards above than below: the manubrium and body of the breast-bone become bent at the angulus Ludovici. But sometimes the spine is much more arched than the sternum, and this may be the case to such an extent, in a thorax highly

Fig. 7.



Bilateral Enlargement of Emphysema.

Inner line = emphysematous chest.
Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line = natural adult chest.
Actual measurements in centimeters:

Circumference = nat. 89· emphys. 87·75
Transverse = ,, 29·6 ,, 27·25
Anteroposterior = ,, 22·25 ,, 25·4

of the flat phthinoid chest but to a higher degree. Phthisis is the disease in which great diminution of both sides of the thorax occurs.

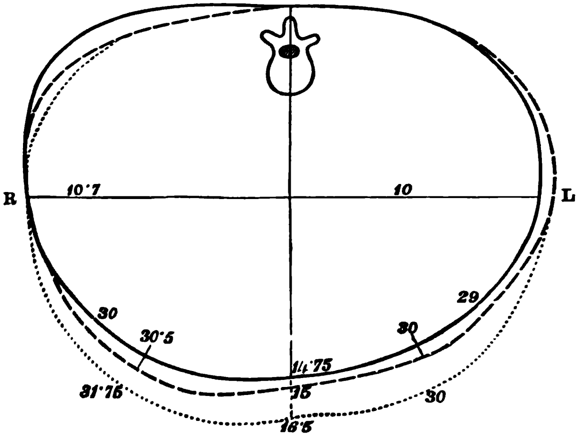
¶ II.—UNILATERAL CHANGES IN SHAPE.

These, like the bilateral, consist in enlargement and diminution.

i. UNILATERAL ENLARGEMENT.—The notes of bilateral enlargement already given are applicable to enlargement of one side only of the chest. The side enlarged, compared with the other, will present these characters : shape rounder ; anteroposterior diameter longer ; length from above downwards diminished, shoulder raised ; spine curved towards unaffected side. I say that the length of the chest from above downwards (the vertical diameter) is diminished, and this is true, provided that the floating ribs be excluded from consideration : let anyone who doubts this statement inject the pleura of a dead subject with air, and watch the changes which ensue. The anteroposterior enlargement becomes very obvious when the physician stands behind the patient so as to look obliquely over his shoulders and the front of his chest. In children the best notion of the enlargement and roundness of the affected side is gained by grasping both sides with the two hands, the thumbs being placed tip to tip upon the spines of the vertebræ. Circumferential measurements of the two sides are often made, but be it remembered, first, that considerable increase in the sectional area of the chest may occur, and the length of the periphery remain the same, by the passage of the elliptical form into the circular : and next that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. Add this consideration, too, that the walls of the healthy side must follow the anteroposterior projection of the diseased side : and then it will be plain why, as a matter of fact, the perimeter of the expanded side often measures very little more, nay even less, than that of the side which is not diseased. The cyrtometer alone, by indicating shape as well as circumference, affords us the true means of recording the amount of a unilateral enlargement.

The causes of unilateral enlargement are increase in the size of the lung or effusion of fluid into the pleura. Increase in the size of one lung occurs in vicarious hypertrophy compensatory of chronic disease whereby the other lung is put out of play: unilateral hypertrophous emphysema, the other lung being healthy, is not found. Effusion of fluid into the pleura, however, causes the greatest enlargement: inflammatory effusion, pneumothorax, extensive hæmothorax. Hydatid disease has the same effect.

Fig. 9.



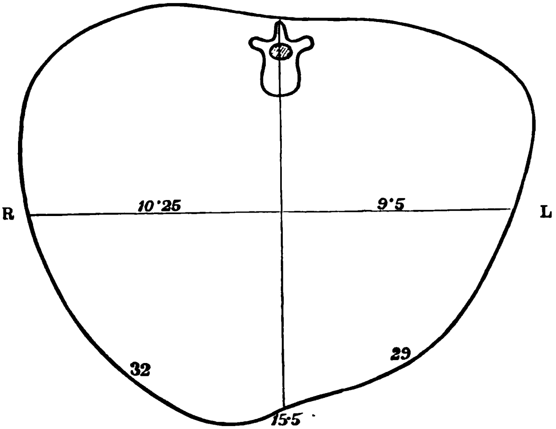
Unilateral enlargement of chest (right side); artificially produced by injecting air into the right pleural cavity.

Unbroken line = outline before injection.
Broken line = outline after moderate distension.
Dotted line = outline after extreme distension.

Figures at bottom of vertical line indicate the anteroposterior diameters: along horizontal line indicate transverse semidiameters: remaining figures indicate right and left semicircumferences.

ii. Unilateral Diminution.—In unilateral shrinking of the chest the circumference and the anteroposterior diameter are diminished; the side looks flat before and behind, having lost its rounded shape and become angular; the ribs are closer together than natural; the shoulder is lower; and the spine curved towards the healthy side. The perimeter of the contracted side is diminished; and, when there is vicarious enlargement of the other lung, the

Fig. 10.

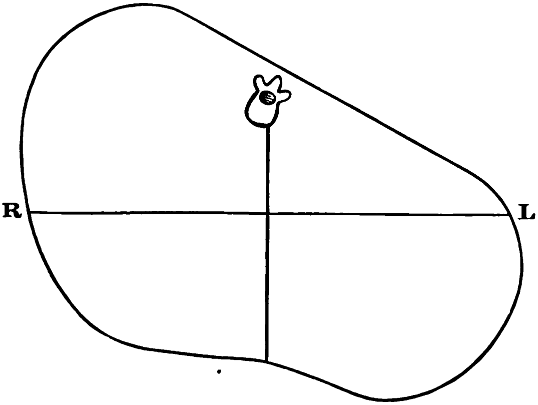


Unilateral retraction of chest: consequent upon cirrhosis of left lung in a girl of fourteen. Figures as in last sketch.

difference between the circumference of the two sides becomes very great.[1]

Unilateral diminution of the chest, when chronic, is commonly attended by an adherent pleura; a condition itself the result of past pleurisy; or connected with phthisis, cirrhosis

Fig. 11.



Shape of Chest in Scoliosis.

From a girl aged eight years and a half. One-third the natural diameter.

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[1] In a case of excessive unilateral contraction of the chest, consequent upon empyema, and associated with distension of the other side, I noted that a very powerful inspiratory expansion and forward movement of the distended side was accompanied with distinct recession and backward movement of the front of the contracted side, as if it were drawn upon and straightened.

or cancer. An acute unilateral shrinking of the chest occasionally occurs in children, as a consequence of collapse of one lung due to an obstruction of the main bronchus.

Scoliosis sometimes induces a shape of chest which, when regarded from the front or from the back alone, strongly simulates unilateral contraction. The cause of this is to be found in the rotation of the vertebræ round their longitudinal axis. The side which looks contracted in front is protuberant behind, and vice versâ.

¶ III.—LOCAL CHANGES IN SHAPE,

namely local bulging and shrinking.

i. LOCAL BULGING.—Bulging of a part of the chest walls is met with in circumscribed pleural effusions, in pericardial effusions, in large phthisical excavations, in hernia of the lung, in hypertrophy and dilatation of the heart, in pointing empyemata, in aneurysms, in cancerous, hydatid, and other tumours.

The bulging indicative of cardiac or pericardiac enlargements occurs between the third and seventh cartilages on the left side, and extends from the left nipple line to the sternum or even to the right nipple line.[1]

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[1] Norman Moore has studied this form of bulging with

ii. Local Shrinking.—Shrinking of a part of the chest walls, when due to intrathoracic disease, is usually attended, like unilateral contraction of the whole chest, by pleural adhesions. Shrinking of the apex of one side is very common in phthisis.

Cup-like Hollow.—A cup-like depression of the lower part of the sternum and of the attached cartilages is not uncommon.[1] The pit is sometimes so broad as to reach up to the level of the third rib, and sometimes so deep that it seems as if the bottom must almost touch the spinal column. This deformity may follow unilateral pleurisy, and yet be perfectly symmetrical; even although one side only of the chest be contracted. Pericardial adhesion, likewise, may cause a hollow of this kind. Hooping cough will do the same. Croup, in infants, is often attended by enormous inspiratory recession of the whole corpus sterni below the angulus Ludovici: whence I infer that the crateriform depression, like the transversely-constricted chest and the lateral contractions of

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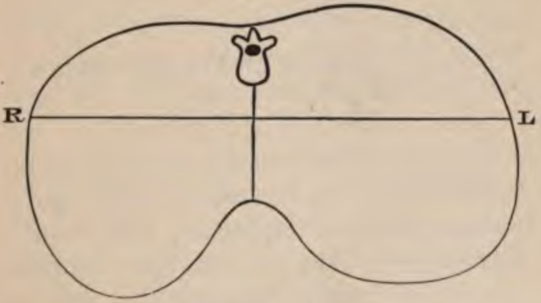
the help of the cyrtometer (Observations on the shape of the chest in cases of hypertrophy of the heart: London, 1873).

[1] In the Museum of St. Bartholomew's Hospital there is a fine skeleton showing this deformity: Case B, No. 3510.

pigeon breast, is mainly dependent upon the same essential condition, namely, obstructed inspiration. A slight degree of a similar deformity is found in many shoemakers : in whom it is due to pressure from without. Graves tells of a young man whose sternum was so yielding that it could be easily pushed back so as to make a cupping such as just described.[1] When the deformity follows hooping cough or pleurisy there is hope that it may disappear in the course of time : but a well-marked depression tends to be permanent.

Fig. 12.



Cup-shaped Depression.

From a man aged eighteen years. Tracing taken on level of xiphoid, and reduced to one-fourth the natural diameter.

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[1] Clinical Lectures. 2nd edit. vol. ii. p. 521.

SECTION II.

MOVEMENTS OF THE CHEST.

These are of three kinds, movements of respiration, movements of the heart, and movements wholly unnatural.

ARTICLE I —MOVEMENTS OF RESPIRATION.

¶ I.—IN HEALTH.

By study of the living thorax in health and disease we learn :—That the diaphragm is the great means of inspiration : That, in quiet breathing, the chief use of the intercostal muscles is to maintain the position of the ribs (or the expansion of the chest) during the descent of the diaphragm : That, when the descent of the diaphragm is hindered, or when inspiration becomes more laboured than natural, the intercostals contract more strongly, so as to dilate the chest by raising the ribs : That, when inspiration becomes as forcible as possible, other muscles, which act by raising the collar bones and first ribs, come into play, namely, the sternomastoids, scaleni, omohyoids, and upper part of the trapezii : That quiet expiration is due to the cessation of all muscular contraction: That forced expiration is performed by means of the abdominal muscles (especially the recti), the latissimi dorsi, and lower part of the trapezii. Add these corollaries:—That the diaphragm and intercostals are antagonist, although they concur to produce one and the same result: That forced inspiration tells upon the upper chest and true ribs: That forced expiration tells upon the lower chest and false ribs. These facts explain, and are illustrated by, much that has been said about the shape of the chest in foregoing pages: and it will now be clear why paralysis of the diaphragm causes bilateral enlargement of the chest, and paralysis of the intercostals, bilateral diminution.

Great is the number of instruments which have been invented for registering the respiratory movements and powers: stethographs, stethometers, thoracometers, spirometers, pulmometers, pneumatometers, anapnographs: a field of study which seems to be uncommonly barren, and to have repaid the outlay of toil and wit thereupon with a very scanty yield to diagnostics.[1]

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[1] Ransome: On Stethometry. London, 1876. Also: Med. Chir. Trans., 1881, vol. 64, p. 185.

¶ II.—IN DISEASE.

Mere increase and mere diminution of respiratory movement, bilateral, unilateral or local, scarcely demand a long notice. Perversions in the character of the movement are more important: they may be arranged under five heads, inspiratory dyspnœa, expiratory dyspnœa, non-expansive inspiration, respiration wholly thoracic, and respiration wholly abdominal.

i. INSPIRATORY DYSPNŒA.—Inspiratory dyspnœa, if unattended by obstruction to the air-draught into the lungs, is made manifest by simply excessive thoracic movement. If, on the other hand, there be such an obstruction, the inspiratory movements tend to be perverted, that is, to differ in kind from the natural movements. But these perverted movements hardly become apparent unless the chest-walls are yielding, as in the case of children: and then we see the expansion or over-expansion of the upper chest, together with that recession of the lower chest below the nipples, which have been so often described and explained already. Perverted movements, such as these, imply obstruction to the air-draught, no matter where the obstruction be. The more yielding the chest, the less the obstruction necessary to cause these perverted movements, and hence they are especially well-marked in infants; indeed, in rickety infants no positive obstruction at all is necessary. Even unilateral diseases, such as empyema and plugging of a bronchus, are sometimes attended in children by these bilateral results of inspiratory dyspnœa, namely, by great and equal recession of the base of the chest on both sides. In obstruction to the larger air-passages the supraclavicular regions usually partake in the recession. Long-standing obstructive inspiratory dyspnœa is the cause of the transversely-constricted, the pigeon-shaped, the rickety and the bilaterally enlarged chests. In the last case, the cervical accessory muscles of inspiration are more or less permanently contracted.

ii. EXPIRATORY DYSPNŒA.—In this condition the expiratory movement is exceedingly laborious and prolonged. Expiratory dyspnœa occurs whenever the lungs are over-distended with air which they cannot expel; as in emphysema, asthma, and bronchitis.

iii. NON-EXPANSIVE INSPIRATION.—The chest-walls are elevated powerfully, yet expanded little or not at all. This sign may occur, on one side or on both, whenever the texture of the lung is impermeable to air, or non-expansible (as in pleurisy with effusion, pneumothorax, dense pleural adhesions, phthisis and cancer), or when the thorax cannot be any more dilated (as in emphysema).

iv. Respiration wholly Thoracic.—The female type of respiration carried to the extreme. Occurs in diseases which interfere with the action of the diaphragm: paralysis of the diaphragm, great pericardial effusion, peritonitis, ascites, abdominal tumours.

v. Respiration wholly Abdominal.—The male or infantine type of respiration carried to the extreme: respiration being performed almost wholly by the diaphragm. Occurs when spinal or nerve disease paralyses the intercostal muscles.

Article II.—Movements of the Heart.

The chief movement of the heart, detected by inspection, is the Impulse. Systolic recession of certain interspaces is sometimes visible.[1]

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[1] Throughout this book, the word "systolic" means contemporaneous with the systole of the ventricles; "diastolic," with their diastole.

¶ I. MOVEMENTS IN HEALTH.

i. The Impulse.—The movement of any part of the heart, which underlies the chest-walls, may be seen and felt in favourable circumstances. But there is commonly only one impulse in health. The part of the heart which strikes the chest-wall appears to be a spot upon the anterior surface of the right ventricle, about three-quarters of an inch above the apex: the apex itself being separated from the thoracic parietes by a layer of lung which is a finger broad.

The Time of the impulse coincides, not with contraction of the ventricles only, but with contraction of the auricles, of the ventricles, and closure of the semilunar valves. Wherefore the impulse is not exactly systolic, but is also præsystolic and postsystolic. Nevertheless it is generally accurate enough to deem the impulse simply systolic.

The Position of the impulse is the fifth left interspace, midway between the nipple and the parasternal lines. In children, the heart frequently lies high, so that the impulse occurs in the fourth interspace, and rather more to the left than in adults, that is to say, in the nipple line. In many old people the heart lies low, so as to beat against the sixth interspace. By a deep inspiration the impulse can be depressed half an inch. By lying upon the left side the position of the impulse shifts to the nipple line or beyond it. By lying upon the right side the position of the impulse becomes uncertain.[1]

The Extent of the visible impulse (the heart beating quietly) is small, not greater than a square inch.

The Force of the impulse is best estimated by palpation.[2]

ii. Recession of the Chest-walls.—Is occasionally perceptible, during the systole, in the third, fourth, or even the fifth intercostal spaces on the left side, close to the sternum: this, especially in persons who are very thin.

¶ II. MOVEMENTS IN DISEASE.

i. The Impulses.—Three kinds of impulse are seen in disease: namely the apex-beat proper, more or less changed in its characters:

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[1] The very uncommon case of transposition of the viscera, and the still more uncommon case of congenital malposition of the heart only, may be alluded to here.

[2] The topic of epigastric pulsation is discussed in chap. 6, sect. 4.

an impulse of some part of the right ventricle above the apex-beat, especially the conus arteriosus ; and an impulse of the right auricle.

a. Impulse of the apex-beat.

The Position of the apex-beat will be changed by whatever enlarges the heart or displaces it.[1]

Enlargement of the left ventricle causes the apex-beat to shift to the left of the left nipple line, in the fifth, sixth, or seventh interspace.

Enlargement of the right ventricle has one or both of two effects. First : the extent of the impulse to the right of the left parasternal line is increased ; for the cardiac enlargement causes the lungs to shrink away, so that the impulse of other parts of the right ventricle than the apex-beat comes to the surface, and is seen and felt. Next : the apex-beat may be displaced to the left; the left ventricle being natural in size.

Displacement of the heart to the left may be so great as to cause the impulse to beat in the axillary line, and in any interspace from the second to the sixth. The causes of this kind of displacement are the following: diseases of the abdomen (ascites, tympanites, tumours) whereby the diaphragm is raised, and the heart comes to lie more horizontally than is natural; effusions of air or liquid into the right pleura; tumours to the right of the heart; retraction of the left lung, whether by phthisis, cirrhosis, collapse or old pleurisy.

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[1] Consult the chapters on the Position of the Diaphragm, and of the Mediastinum, with reference to displacements of the heart.

Displacement of the heart to the right may cause the impulse to be seen anywhere to the left of the right nipple line, in any interspace from the fourth to the seventh, or in the epigastrium. The causes are similar to those of displacement to the left, excepting abdominal enlargement. And, whatever may be the case in dislocations of the heart rapidly produced, there is no doubt that chronic disease can displace the heart so that its very apex-beat shall be felt in the right nipple line.

β. Impulse of the right conus arteriosus.

Yet when the impulse of a diseased or displaced heart is seen to the right of the natural position, it is often some part of the ventricle, other than the apex, which strikes against the chest; and this part is usually the conus arteriosus. A white patch, telling of long friction, is often seen at this spot after death.

γ. Impulse of the right auricle.

A systolic impulse seen in the fifth interspace and the right parasternal line may be due to a dilated right auricle.[1]

The Extent of the impulse (meaning thereby visible or palpable contact of the heart with the chest-wall) may be increased; and then the movement sometimes takes on a fluctuating or peristaltic character. The causes of a too extensive impulse are these: enlargement of the heart, the lung being pushed aside; shrinking of the lung; and whatever presses the heart against the chest-wall, for instance a narrow chest, or a tumour in the posterior mediastinum, especially aneurysm of the descending aorta.

ii. RECESSION OF THE CHEST-WALL.—Recession of the chest above the apex-beat is much more frequently met with in hypertrophy of the heart than in health. The apex-beat itself is sometimes strongly dimpled inwards during the systole; a sign which was formerly thought to indicate the presence of internal and external pericardial adhesion ; but which is now believed to be of small value.

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[1] Markham: Med. Chir. Trans. vol. xl. 1857. In this case the impulse was systolic, and the walls of the auricle were almost destitute of muscular fibre; so that the most probable cause of the impulse was a sudden filling of the auricle with blood during the ventricular contraction. An aneurysm of the ascending aorta may beat in exactly the same situation.

ARTICLE III.—MOVEMENTS WHOLLY UNNATURAL.

Movements, that is to say, which have no counterpart in the healthy state. The pulsation of aneurysmal tumours is of this kind.

> "More faithful witnesses are eyes than ears."
> HERACLITUS : FRAGMENT XV.

CHAPTER III.

PALPATION.

PALPATION, or application of the hand to the surface of the chest, is often useful as a means of confirming notions acquired by the eye concerning the shape, size, or amount of movement of any part of the thorax. But palpation has an independent value, and can detect thrills and impulses which are imperceptible to the eye. These signs will be considered in order, as they relate to the lungs and pleuræ, the heart and pericardium, and the large bloodvessels.

SECTION I.

LUNGS AND PLEURÆ.

ARTICLE I.—VOCAL THRILL.

¶ I.—IN HEALTH.

When a person speaks, a quivering or vibration may often be felt upon the surface of the chest: the vocal thrill. The strength of thrill is in direct proportion to the depth and loudness of the voice, and therefore is best marked in adult men, and is often absent in women and children. The reason of this seems to be that only in low-pitched notes are the vibrations sufficiently far apart to be perceptible to the hand. The thrill is conducted from the larynx both by the tissues and by the air in the air-passages: it is strongest over the lungs, and is weakened as we pass away from them: and it is naturally more intense on the right side than on the left.

In examining vocal thrill it is best to make all patients repeat the same sound: the words "ninety-nine" said in a low tone are very suitable for the purpose.

¶ II.—IN DISEASE.

Vocal thrill is diminished or abolished by whatever separates the lung from the chest-wall, and by whatever renders the lung quite impermeable to air. An effusion into the pleura, whether of liquid[1] or gas, is a common cause of loss of vocal thrill; but, unless the effusion be great, the thrill is not wholly abolished, being somewhat conducted by the thoracic walls. Solidification of the lung does not much diminish vocal thrill, unless the solidification be very dense. Wherefore ordinary pneumonic and phthisical consolidations do not lessen the thrill, and may even increase it; but pneumonic exudation which packs the lung hard, massive phthisical solidification, and cancer, these abolish vocal thrill, unless indeed a large bronchus be in intimate connection with the solid mass.

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[1] This fact seems to have been discovered by Reynaud: see his memoir quoted under the head of Pleural Friction Sound. Stokes (Diseases of Chest, p. 497) refers to Reynaud's Inaugural Thesis published in 1819, which I have not seen.

Undoubted increase of vocal thrill is an uncommon and unimportant sign.

The thrills produced by cough and rales are of no practical value.

ARTICLE II.—PLEURAL FRICTION:

May occasionally be felt, but it cannot be easily distinguished from rhonchal thrill, except by means of auscultation.

ARTICLE III.—FLUCTUATION.

Fluctuation of a liquid effusion in the pleura may sometimes occur.

i. Fluctuation produced by striking the chest is an uncommon, and unnecessary, sign, inasmuch as a diagnosis will have been attained long before the disease has gone so far as to yield the sign of fluctuation. When the intercostal spaces are widened and rendered tense, fluctuation may sometimes be produced and felt by two fingers placed far apart in the same interspace. The splash of hydropneumothorax may be sometimes felt, but is much better heard.

ii. Fluctuation of a pleural liquid effusion is occasionally produced by the action of the heart (see the chapter on empyema).

SECTION II.

HEART AND PERICARDIUM.

ARTICLE I.—IMPULSE.

¶ I.—SYSTOLIC IMPULSES.

Palpation will serve to confirm the information yielded by the eye as to the position and extent of impulses of the heart against the chest-wall. Sometimes impulse is palpable when it is not visible.

Palpation, moreover, enables us to say whether the force of impulse is weaker or stronger than natural.

i. The force of impulse is diminished by whatever weakens the heart, and by whatever separates it from the surface of the chest-wall.

ii. The force of impulse when increased, that is to say, heaving impulse, is much better appreciated by applying the stethoscope and head to the apex-beat, than by the hand alone.[1] Knowledge of this fact led to the discovery of auscultation. When heaving impulse is well marked, it is able to overcome the greatest pressure which the patient can bear, and may be regarded as a sure and certain sign of cardiac hypertrophy. It is seldom difficult to distinguish the knocking of a palpitating heart from true heaving.[2]

The Cardiograph (an instrument invented by Chauveau and Marey,[3] whereby the movements of a cardiac impulse may be registered) has confirmed or revealed the truth of the following propositions :—

That the contraction of the auricles immediately precedes that of the ventricles : That the systole of both ventricles is exactly synchronous: That the impulse coincides with the contraction of the auricles and ventricles, and the closure of the semilunar valves: That the second sound follows immediately upon the ventricular systole: That the auriculo-ventricular valves vibrate during the greater part of the ventricular systole: That the closure of the aortic semilunars precedes that of the pulmonary semilunars.

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[1] Laennec : Auscult. médiate. 2nd edit. vol. ii. p. 394.

[2] Laennec : ibidem, vol. ii. p. 397.

[3] Chauveau et Marey ; Mém. de l'Acad. de Méd. : Paris, 1861.

¶ II.—DIASTOLIC IMPULSES.

Diastolic cardiac impulses are of two kinds:

i. A diastolic impulse (the back stroke, as it was called by Hope[1]) can sometimes be felt. It most frequently occurs with hypertrophied hearts, and is probably due to sudden diastole of the ventricles, after a powerful contraction.

ii. Sudden closure of the pulmonary sigmoid valves sometimes causes a sharp invisible diastolic impulse, felt at the second left interspace close to the sternum, or at the apex-beat. This sign indicates a loud second sound, and is due to the same conditions.

¶ III.—PRÆSYSTOLIC IMPULSE.

It is said that contraction of the auricles, when they are much hypertrophied, may give rise to a short præsystolic impulse above the fourth cartilage, and either to the right or to the left of the sternum. An auricular impulse which is systolic, the auricle being passive at the time, has already been referred to (p. 43).

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[1] Diseases of the heart: 3rd edit. pp. 67, 272.

ARTICLE II.—VALVULAR THRILLS.

Valvular thrill is a quivering or vibration felt by the hand applied to the region of the heart in certain forms of disease. Its likeness to the purring of a cat led Laennec[1] to invent the name "frémissement cataire" for the phænomenon; by its discoverer, Corvisart, it had been described before under the term "bruissement."[2] A thrill must be well marked to be of value as a physical sign: what may be considered a well-marked thrill can be learnt by experience alone.

Thrills are palpable murmurs, and are due to the same physical condition; that is to say, to vibration set up by fluid in motion. Vibration must be strong and slow in order to be palpable. The solids also must be apt for conduction; in other words, apt for convibration; they must be elastic, tolerably homogeneous, and of a certain mean tension. Along fluid currents, thrills are conducted according to the laws of murmurs.[1]

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[1] Traité de l'auscultation médiate et des maladies des poumons et du cœur. 2nd edit. vol. ii. p. 448. Paris, 1826.

[2] Essai sur les maladies et lésions organiques du cœur et des gros vaisseaux. 2nd edit. p. 232. Paris, 1811.

Thrills, like murmurs, are of many kinds, and are distinguished by determining the place and time at which the thrill is felt.

i. Thrill felt in the second right intercostal space, close to the sternum, or at the episternal notch: indicative of a lesion at the aortic orifice. Systolic, the orifice is obstructed: diastolic, the valves are incompetent.

ii. Thrill felt in the second left intercostal space, or over the third left costal cartilage, close to the sternum: indicative of a lesion at the pulmonary orifice. Systolic, the orifice is obstructed: diastolic, the valves are incompetent. Here, however, it must be noted that, in aortic regurgitation, the heart may be displaced so much to the left, that diastolic thrill, produced at the aortic orifice, shall be felt to the left of the sternum, in the second and third interspaces.

iii. Thrill felt at the apex-beat: indicative of a lesion at the mitral orifice. Systolic (not a mere quivering impulse, but a thorough thrill), the valves are incompetent; præsystolic (running up into the impulse, and brought to an end thereby), or diastolic (alternating with the impulse), the orifice is obstructed.

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[1] Refer to the article on Murmurs in general, and to that on the conduction of Percussion sounds.

iv. Thrill felt over the fourth left costal cartilage, or in the fourth left interspace, close to the sternum: indicative of a lesion at the tricuspid orifice. Præsystolic, the orifice is obstructed.[1]

Concerning the explanation of the place and time of these many thrills, the reader may refer to the chapter on Endocardial Murmurs: all that is said on these topics with regard to the murmurs, applies to the corresponding thrills. It may be well to remark that thrill felt to the right of the sternum is often due to aortic aneurysm.

ARTICLE III.—PERICARDIAL FRICTION.

The rubbing of pericardial exudations is sometimes palpable: a matter of small practical value. Friction is usually very unlike valvular thrill.

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[1] Gairdner: Edin. Med. Journal: v. 871. Feb. 1860.
Haldane: Edin. Med. Journal: x. 271. Sept. 1864.

Aortic aneurysm, which opens into the pulmonary artery, may cause thrill in this place: see Wade's paper referred to in chap. v.

ARTICLE IV.—FLUCTUATION.

In chronic copious pericardial effusions fluctuation may sometimes be felt, or even seen: an observation as old as Senac,[1] if not as old as Galen.[2]

SECTION III.

LARGE VESSELS.

Impulses and thrills produced in the large bloodvessels will be considered in the chapters on Thoracic Aneurysms.

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[1] Traité de la structure du cœur. 2nd edit. vol. ii. p. 364. 1783. In Senac's patient, the fluctuation occurred during palpitations, in the third, fourth, and fifth interspaces.

[2] De locis affect. lib. v. cap. 3.

"Modo pectora præbet
[Sollerti] palpanda manu."
OVID. METAM. ii. 866.

CHAPTER IV

PERCUSSION

SECTION I.

INTRODUCTORY.

ARTICLE I.—HISTORICAL.

PERCUSSION, or the art of striking a part of the body so as to beget a sound useful for the discovery of disease, has been practised from the earliest times. Employed at first in the diagnosis of abdominal diseases (to distinguish tympanites from ascites [1]), it was not until the middle of the last century that percussion was applied to the discrimination of pectoral diseases. This important extension of the powers of percussion we owe to Auenbrugger, who in 1761 published a small book descriptive of his method.[1] He tells us that he was prepared to suffer from envy, hatred, and calumny; we know that he endured what is harder to bear, simple neglect.[2] For many years after the discovery, Stoll alone acknowledged its value;[3] but it was Corvisart who, guided by his favourite author, Stoll, may be said to have discovered Auenbrugger. For twenty years Corvisart practised percussion, and finally, in 1808, he published a translation of the Inventum Novum.[4] Henceforth the importance of percussion was admitted by all.

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[1] This means of diagnosis is probably at least as ancient as Hippocrates. During the three busy centuries after Hippocrates, the Greeks invented the word tympanites, which was in familiar use by the time of Celsus.

The principles of the Inventum Novum are two: first, that the sounds produced by percussion must be regarded simply as acoustic phænomena, and named accordingly; secondly, that the sounds are to be explained by reference to corresponding physical states, that is to say, to the presence or absence of air in the part percussed.[1] Auenbrugger's doctrines remained unassailed until Piorry, in 1826,[2] sought to establish a new theory founded upon the principle that every organ yields to percussion a peculiar sound. Hence, a series of typical sounds: femoral, jecoral, cardial, pulmonal, intestinal, stomachal, osteal, humoral, and hydatid.[3] This scheme was afterwards discarded by its author, who proposed another in its stead: we will not forget that Piorry invented the pleximeter. In 1839, Skoda[4] restored Auenbrugger's principles, saying that "we must first determine every possible variety of percussion-sound, and ascertain the conditions on which each variety depends; and then endeavour to reconcile our observations with the well-ascertained laws of sound." Wherefore, in the first place, Skoda[1] adopts Auenbrugger's principle of nomenclature, and distinguishes percussion-sounds according as they are full or leer, clear or dull, tympanitic or not tympanitic, high or low; terms which will be explained hereafter. In the next place, Skoda developes Auenbrugger's second principle by maintaining that "the different sounds, which percussion produces over the regions of the liver, the spleen, the heart, the lungs, and the stomach, do not depend upon any peculiarities in these organs, but upon variations in the quantity, distribution, and tension of the air, present in the regions in which they lie, and upon the force of the percussion stroke." The doctrines of Auenbrugger are in essence the doctrines of the present day.

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[1] Leopoldi Auenbrugger, M.D., Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi. Vindobonæ, 1761. Translated by Forbes: Original cases, &c. London, 1824.

[2] Leopold Auenbrugger, der Erfinder der Percussion des Brustkorbes, und sein Inventum Novum: von Prof. Dr. Clar. Graz, 1867.

[3] Stoll: Rationis medendi pars tertia: p. 155. 1780. Prælectiones in diversos morbos chronicos: vol. i. p. 86: 1788.

[4] Nouvelle méthode pour reconnaître les maladies internes de la poitrine, par la percussion de cette cavité par Avenbrugger, ouvrage traduit du latin et commenté par J. N. Corvisart. Paris, 1808.

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[1] Auenbrugger: §§ 17, 18, scholia.

[2] Paper read before the Parisian Academy of Medicine in that year.

[3] De la percussion médiate et des signes obtenus à l'aide de ce nouveau moyen d'exploration, dans les maladies des organes thoraciques et abdominaux. Paris, 1828. Page 33.

[4] Abhandlung über Perkussion und Auskultation Wien, 1839. This work reached a sixth edition in 1864. A translation of the fourth edition, by Dr. Markham, was published in 1853.

ARTICLE II.—METHOD OF PERCUSSION.

i. IMMEDIATE PERCUSSION.—This was the method employed by Auenbrugger, and may be described in his own words: "Let the chest be percussed by the tips of the fingers drawn together side by side and stretched out straight; let the chest be covered by a vesture, or the hand by a glove (not of smooth leather), for if the naked chest be struck by the naked hand a smack ensues which hides the character of the sound we wish to produce."[1] Corvisart often percussed by the whole flat of the hand. At the present day immediate percussion is practised only upon the clavicles (they constituting pleximeters), or when the physician wishes to obtain a rough preliminary notion of the resonance of a considerable extent of the chest surface.

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[1] Skoda: Markham's trans. pp. 4 and 6.

ii. MEDIATE PERCUSSION. — Auenbrugger's glove was obviously an approach to that mediate percussion which was first systematically practised by Piorry.[2] Piorry interposed a thin plate of ivory (pleximeter) between the chest and the percussing fingers; the fingers moreover he kept half bent, so that percussion was made by their very tips, and not by their pulps, as in Auenbrugger's process. To substitute a hammer for the percussing fingers was no great stretch of inventive genius: Laennec frequently percussed with his massive stethoscope: from time to time divers plessors have been contrived, which may be seen in the shops of surgical instrument makers. But hammers and ivory pleximeters cannot feel, and feeling has much to do in percussion; wherefore few physicians employ any instruments, except the fingers.[1] The movement of the percussing hand should proceed from the wrist and not from the elbow or shoulder, a nicety in manipulation which is acquired by practice.[2]

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[1] Inventum novum, §§ 4, 5.

[2] Percussion médiate, p. 14.

ARTICLE III.—THEORY OF PERCUSSION.

The first object of percussion is to discover what kind of sound is emitted by the part percussed. A secondary object is to discover the degree of resistence, or the density of the stricken spot.

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[1] Piorry. Traité de plessimétrisme et d'organographisme. Paris, 1866. Here the reader will find the argument stated in favour of the pleximeter. In the same book there is a description of divers plessigraphs.

Wintrich: Einleitung (Virchow's Handbuch der spec. Path. und Ther.: 1854), p. 5. Here the cause of the hammer is pleaded.

[2] For the use of resonators and sensitive flames in examination of the chest, see Gerhardt: Lehrbuch der Aus. und Perc. 3rd edit. 1876; pp. 81, 116, 161, 272.

For tuning forks, see Wintrich: Einleitung: p. 44 And Baas: Arch. für klin. Med. 1872, vol. xi. p. 9.

¶ I.—PERCUSSION SOUNDS.

All sounds own the common properties of loudness and duration: two other properties, namely, tone and pitch, are peculiar to some sounds only.[1]

The first division of percussion-sounds depends on tone; its presence or its absence. Some sounds are tones and some are not; hence the main distinction. And also the most ancient distinction; whereof the appreciation was, as we have seen, the first step taken in the art of percussion: the difference between a tone and a noise which is not a tone, being the difference between the percussion-sound of a tympanitic and of an ascitic belly.

CLASS I.—PERCUSSION-TONES.

Percussion-tones are also called clear sounds or resonances, for reasons which will appear hereafter. Percussion-tones shall be considered under the sundry headings of their four common properties, namely, their loudness, duration, pitch, and tone.

I. Loudness.—Loudness is not a character of much practical worth, depending as it does, not only upon the conditions of the sonorous material, but also upon the force of the percussion-stroke.

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[1] Tyndall: Sound. 1867, p. 69.

II. Duration.—Duration is a somewhat more important quality. The prime property assigned by Skoda[1] to a percussion-sound, its fulness or its leerness[2] (ideas adopted from Laennec[3]) is, in fact, a compound perception, made up chiefly by the duration of the sound.

III. Pitch. — Pitch is a more important quality. The many different degrees of pitch, distinguishable in percussion-tones, are reduced to three or four heads, which have received arbitrary names. So that we have a kind of scale of percussion-tones.

i. The lowest-pitched tones are called Tympanitic,[4] because they are usually yielded by a tympanitic belly.

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[1] Markham's translation: p. 8.

[2] Skoda's word "leer" is translated by Markham "empty": I formerly suggested "scanty." But, indeed, the word "leer" needs no translation, for it is English as well as German, and bears the same meaning in both tongues. See Halliwell's Archaic Dictionary: Leer or Lear.

[3] Auscultation médiate: vol. i. p. 28.

[4] Skoda gives the word "tympanitic" quite another meaning. So well as I can make out, tympanitic means, with him, the same thing as that which I call clearness of tone, or rather perfect clearness. A tone, which is

ii. The somewhat more highly-pitched tones I have proposed to call Sub-tympanitic, thereby to keep up the traditions of Auenbrugger;[1] it is the note usually yielded by healthy lungs, in their natural state of distension; the Pulmonal note of Piorry.

iii. The still more highly-pitched tones are called Tracheal[2] or Tubular, being more or less like the note yielded by the trachea, when the mouth is a little open.

iv. The highest-pitched tones are called Osteal,[3] because they are yielded by the hard solid tissues, cartilage and bone.

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muffled to any degree, is "not-tympanitic" with Skoda. Wintrich makes this complaint: "Ich muss offen gestehen, dass mir die Expositionen des Wiener Meisters über alle die genannten Schalldifferenzen ganz unklar geblieben sind, zumal wenn der Versuch gemacht wurde, sie mit den Gesetzen der Physik oder auch nur den praktischen Auffassungen der Musiker in Verbindung zu bringen." Einleitung, p. 39. French disciples of Skoda call his tympanitic note, le bruit skodique: if they mean hereby that Skoda was the first to draw attention to the sound, they are very much mistaken.

[1] Sonus, quem thorax edit, talis observatur, qualis in tympanis esse solet, dum panno vel alio tegmine ex lana crassiori facto, obtecta sunt. Inventum novum: § 2.

[2] C. J. B. Williams: Lond. Med. Gaz. vol. xxi. p. 918, and vol. xxii. p. 8. 1838.

[3] Piorry: Percussion médiate, p. 31.

IV. TONE.—Thus far I have treated of percussion-tones as if they were highly gifted with that property; but such is not the case. Indeed, they are seldom pure or clear; their musical quality is mostly impaired; the tone is obscured, muffled, or dulled. And there are all degrees of tone-dulling down to utter dulness, or a noise wholly bereft of tone. Wherefore percussion-sounds are said to be more or less clear, more or less dull. And here I would affirm that these words, clearness and dulness, relating to the degree of purity of tone, are not only hallowed by long usage, but also possess a strict scientific meaning.[1] We inherit them from Auenbrugger,[2] who, in a sentence of fourteen words, has summed up the acoustic phenomena of percussion. The sound is a tone, clear or muffled, even to complete privation; this is the first and great distinction. And next, the tone is of a pitch higher or lower. Upon these two hang the whole theory and practice of percussion.

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[1] Tyndall: Sound, p. 51, for example.

[2] Sonitus vel altior, vel profundior; vel clarior, vel obscurior, vel quandoque prope suffocatus deprehenditur. Inventum novum; § 10, scholium. Curiously misunderstood by Corvisart: Nouvelle méthode, pp. 30, 31.

CLASS II.—PERCUSSION-NOISES.

Concerning the noises of percussion, the sounds which are toneless or dull, there is but little further to say. They possess the qualities of loudness and duration; but these, in percussion, are lightly esteemed. Any other differences depend upon the degree of dulness, whether absolute or not; whether, in fact, some amount of tone is mingled with it or not.

CLASS III.—BY-SOUNDS OF PERCUSSION.

Certain by-sounds, commonly called metallic, sometimes accompany the sounds which have been described. Metallic sounds are two: metallic ring and cracked pot.

I. THE METALLIC RING (empty bottle note, amphoric note) consists in an overtone[1] superadded to the fundamental tone, which itself may be either clear or muffled. Percussion of the stomach when distended with air will yield the metallic ring. Also fillipping the cheeks sharply, when they are blown out to a degree which may be learnt on trial.

II. THE CRACKED POT sound (metallic chink, cracked metal sound) was discovered by Laennec.[1] It may be likened to the chinking of money, or to the sound produced by clasping the hands loosely together, and striking the back of one of them upon the knee. Percussion of the chest of a healthy screaming baby will often give a cracked-pot sound. A heavy blow, expiration, and the mouth open, are mostly needful for bringing out the chink. Its causes are not always the same ; they will be set forth hereafter.

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[1] Tyndall: Sound, pp. 116 et seq.

II.—PHYSICAL CONDITIONS OF PERCUSSION-SOUNDS.

We have now to examine the differing conditions which underlie the different sounds which percussion of the chest produces : in other words, we have to explain those sounds so far as we can. And, inasmuch as the prime distinction between them lies in the presence or absence of tone, we will begin with the discussion of percussion-tones.

I. Percussion-Tones.—The only tones, with which percussion has to do, are those produced by the vibration of bone, cartilage, or stretched membrane.

The sound of percussed bone and cartilage

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[1] Auscultation médiate : vol. i. pp. 100, 655.

(for instance, that yielded by percussing the skull or shin with the tips of the fingers) partakes from the first of the nature of a tone.

But with stretched membrane the case is different. Percussed in open air, membrane yields no tone. Nor can it be made to do so, unless it vibrate over an air-containing cavity with smooth walls. Under these circumstances, the sound-waves, which the air conducts from the membrane, are stopped and reflected by the walls of the cavity. But reflection alone will not beget a tone, unless the reflection be rhythmical. And rhythmical reflection requires a certain fixed relation between the rate at which the membrane vibrates, and the length of the air-column beneath. Given these necessary conditions, then the vibrations of the membrane become rhythmically reinforced by reflection ; and rhythmical vibration, if rapid enough, will yield a sound having the characters proper to tone. The reinforcements or production of tone by rhythmical reflection is called Resonance.[1]

In the case, then, of a membrane stretched over a cavity containing air, when we strike the membrane we produce a flutter of the air contained in the cavity; and some pulse of this flutter, corresponding to the size of the cavity according to the law just laid down, is raised by resonance to the dignity of a tone.[1] And this is the reason why the words tone and resonance, as applied to percussion-sounds have come to mean the same thing; the only tones which percussion knows (those of bone and cartilage excepted) are produced by resonance.

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[1] Tyndall : Sound, p. 172.

The conditions at which we have now arrived are these: a sac containing air. To yield Tone the sac must be of a certain size. Greater precision than this is unnecessary, and indeed impossible, in the case of the human body, because the tone depends on many conditions other than the mere size of the cavity. The Pitch of the tone, so far as the cavity alone is concerned, is low in direct proportion to the length of the air column.

Let us now apply these doctrines to explain the percussion-note of pneumothorax, which alone fulfils the conditions of a cavity, large, closed and containing air. Were these the only conditions of the case it would be simple enough. But the loudness and clearness of the sound produced depend more upon the sac-wall than upon the bulk of air contained therein. In order to yield tone, the wall must be apt to vibrate rhythmically together with the contained air. In other words a unison vibration, convibration, or consonance of the wall is required to the production of tone. Mere reflection of air-vibrations from the inner surface of the sac is not enough; the production of resonance requires a consonance of the containing membrane, whereby to keep up the vibrations of the air. When we search into the conditions which favour or oppose the convibration of the sac-wall, we find that its elasticity is the chief of them. The tension of the wall is indeed a matter of some consequence; for the membrane must be flexible enough to vibrate rhythmically, and high tension opposes flexibility: a fact which may be demonstrated upon the cheeks, and which is often demonstrated for us by the membrana tympani,—a certain mean tension being needful to good vibration. But the elasticity of the sac-wall is a more important condition than is the tension; inelastic membrane cannot vibrate rhythmically. In sum: the sac-walls must be flexible and vibratile enough for the column of air within to be able to bend them to its own vibration, and thereby to make them its own sounding-board and tuning-fork;[1] else a clear-toned resonance is impossible. When the sac-wall consonates imperfectly the tone is proportionally muffled; and of muffling there are all degrees up to absolute dulness. But the thick, soft and heterogeneous walls of the chest are obviously very far from fulfilling the conditions necessary for the production of a clear tone; and hence the note yielded by percussion of a pneumothorax is more or less muffled.

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[1] Tyndall: Sound, p. 178.

The sound thus generated has to be transferred to the outer air, and the same conditions which make the chest-walls inapt for the production of tone, make them inapt for its conduction. That the chest-walls conduct badly, hardly needs to be proved: but were proof necessary, it would be afforded by the fact that the metallic ring (p. 65), which the percussion-tone of pneumothorax usually possesses, is not heard except by auscultation or putting the ear to the chest. This amphoric or metallic ring is due to an over-tone produced together with the prime fundamental tone: the chest-wall conducts the prime tone to the outer air moderately well, but the metallic over-tone not at all.

Passing from the walls and cavity of the chest to its contents, we have now to consider the percussion-sound afforded by lungs. We have to explain these facts: That the percussion-note of the chest is much the same whether its cavity be occupied by lung in its natural expanded condition, or whether the cavity be occupied, but not distended, by air alone: That the percussion-note of healthy lung is much the same, whether it be percussed when within the thoracic cavity, or when taken out of the body, the natural distension of the lung being kept up: That the percussion-note of lung which has been allowed to relax, contract, lose its distension, is clearer and of higher pitch than the note of naturally distended lung. The principles already laid down (pages 67, 68, 69) are adequate to explain these three main facts, if we assume that the lung, under percussion, vibrates and resounds as a whole, that is to say, like a single sac containing air. The close resemblance between the percussion-sound of distended lung and of undistended pneumothorax justifies this assumption. The pulmonary tissue is immersed in air; when the two vibrate in unison they yield a clear tone; but when the pulmonary membranes do not keep time with the vibration of the air which they enclose, there is a corresponding want of clear tone. Now, the aptitude of elastic membranes for unison vibration is proportionate to their tension; a certain mean degree of tension being necessary for the production and conduction of clear tone; any variation from this degree of tension, more or less, impairs the tone. The heightened pitch of the tone yielded by relaxed lung is due to its diminished bulk.

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[1] Tyndall: Sound, p. 175.

II. PERCUSSION-NOISES.—Privation of the conditions, which have been declared necessary to the production and conduction of percussion-tones, results in percussion-tonelessness, noise, or dulness of sound. The physical conditions of percussion dulness are these: there may be no tone-producing material in the part percussed, or the tone produced may be badly conducted, or the percussion-blow may be badly conducted. With regard to percussion-dulness of lung, in particular, either the resonant parts are rendered less resonant, or the conducting parts less apt for conduction, and the former is by far the more important condition.

III. METALLIC BY-SOUNDS.—The nature of the Metallic Ring has been already explained (p. 65). It is believed to be necessary to the production of this by-sound, that the resonating cavity possess smooth curved walls, and that it be not less than two inches in diameter.[1]

It remains to say a few words upon the Cracked-Pot sound. Its physical conditions are not always the same. i. One kind, that which percussion produces in screaming babies, is believed by Wintrich[2] to take place in the glottis, when the quick and hard blow suddenly sends compressed air between the tense vocal cords, whereby they are thrown into irregular vibrations. ii. In like manner the cracked-pot sound which heavy percussion, upon yielding chests, produces in healthy lungs during mere expiration, is probably glottidean. iii. Another kind is but a variety of the metallic ring, and is associated with it, being produced, in air-containing cavities not wholly closed, when percussion causes a sudden condensation of the air, and so jars the tone. The mouth of the cavity enables the pressure within and without to be quickly equalised. To shut the mouth and nose prevents the production of a cracked-pot sound, but the metallic ring remains.[3] iv. It is likely that the sudden rush of air, through the opening spoken of, is attended by a hiss which heightens the effect of the cracked-pot sound. v. A sound, practically indistinguishable from the cracked-pot sound, is that to which Piorry has given the name of "humoral,"[1] and which is sometimes produced by percussion over cavities which contain both liquid and air; the sound in question being a sort of percussion-rale, due to splashing of the liquid. A bladder, holding both air and water, if strongly percussed just below the surface of the water, yields a humoral sound.

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[1] Six centimeters (nearly two inches and a half): Wintrich: Einleitung, p. 34. If the cavity communicate with a bronchus, the size may most likely be less.

[2] Einleitung, p. 36.

[3] Walshe: Diseases of lungs. 3rd edit. p. 80.

¶ III.—PERCUSSION RESISTENCE.

The sense of resistence felt by the percussing fingers is greater or less in proportion to the greater or less compressibility of the part percussed. Hence solids and liquids are very resistent; air-containing parts much less so. It is by this means that a person percussing learns more from his percussion than does a bystander. Corvisart first drew attention to this sign; Piorry, working out the subject, at last almost came to exalt the tactile sensations of percussion above the acoustic. But, apart from all exaggeration, there is no doubt that the sense of resistence not only re-inforces, as we may say, of the sense want of tone, but also enables us to distinguish between certain states which agree in yielding absolute dulness to percussion. The great resistence of a liquid effusion, and the change which we often perceive on passing from the heart to the liver, are examples of this truth.

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[1] Percussion médiate, p. 31.

Percussion Thrill. A peculiar quivering sensation, called by Piorry the hydatid thrill,[1] is sometimes produced by percussion. The finger feels as if it were repelled several times in succession by a sort of elastic resistence or fluctuation. Most likely the only physical conditions needful to the production of this thrill are an elastic sac tightly full of thin liquid ; conditions fulfilled by hydatid cysts. A stomach filled with water, hung up, and percussed, will yield a thrill : but whether hydatid thrill is ever felt on percussion of the chest, is very doubtful.

Superficial and Deep Percussion. Before proceeding to apply these principles to the practice of the percussion of the chest in health and disease, it will be proper to say a few words upon what are called Superficial and Deep percussion. By progressively increasing the force of the blow from the gentlest tap to the hardest the patient can bear, we influence progressively deeper layers of the part percussed. For example: gentle percussion will elicit as clear a pulmonary note an inch or two below the right nipple as above the nipple; but hard, heavy percussion will produce a tone which is much less long and loud in the former than in the latter spot, the true explanation being this: not that deep percussion brings out hepatic dulness below the nipple, but that, while gentle percussion influences only a small depth of lung, which depth exists as well below the nipple as above it, heavy percussion, influencing a much greater depth of lung, shows, by a marked difference in the length and loudness of the sounds, that a certain thickness of lung does not exist over the liver but does exist above it.

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[1] Son hydatique : Percussion médiate, p. 32.

SECTION II.

PERCUSSION OF THE CHEST IN HEALTH.

The pulmonary, cardiac, and mediastinal regions must be separately considered.

ARTICLE I.—THE PULMONARY REGION.

¶ I.—PULMONARY RESONANCE AND RESISTENCE.

i. TYPICAL.—Frequent experiment upon the healthy chest is the means by which to fix in the mind an idea of the sound and resistence afforded by percussion of the pulmonary regions. When disease affects one side only we possess in the other side a standard of health to which we may refer; even when both lungs are affected, but one more than the other, comparison is still useful: wherefore, to contrast the same parts of the two sides of the chest becomes an important rule in the practice of percussion.

ii. SUB-TYPICAL.—But certain deviations from the type are compatible with a state of perfect health; deviations for the most part due to the thoracic walls. The sound produced by percussion over the sternum, clavicles, ribs, and scapular spines, partakes of the osteal character. Ossification of the cartilages produces the same effect. The greater the quantity of soft tissue, muscular or adipose, which covers the thorax, the greater the muffling of the sub-tympanitic resonance. Hence the percussion-note of the chest is clearer in front and at the sides than behind, clearer in thin persons than in fat; it is sometimes almost impossible to get any sound deserving the name of resonant by percussing the backs of fat, flabby people. When the chest-walls are yielding, heavy percussion of the front of the chest will produce the cracked-pot sound. During the long deep expiration which attends coughing or screaming, the chests of children become much less resonant than natural. Any part of the chest-walls which is stiffly arched bears off the force of the percussion-blow from the underlying organs, and thus weakens more or less the impulse which we wish to impart to them.

¶ II.—EXTENT OF PULMONARY REGION.

The region which yields a pulmonary note extends from the very apex of the thorax on each side, as low as the sixth rib in front, the seventh at the sides, and the tenth or eleventh behind. But sundry viscera encroach upon these limits.

(i.) The Heart causes a certain extent of non-resonance in the anterior part of the chest; see next page. (ii.) The Liver can, by hard percussion, be detected on the right side as high as the fifth or even the fourth intercostal space in front, and ninth or tenth rib behind. (iii.) The

Spleen on the left half of the chest, below the sixth rib laterally, modifies the percussion-sound. (iv.) The Stomach, especially when distended with gas, affords its own resonance to percussion of the lower part of the left side of the thorax as high, it may be, as the fourth rib, in the lateral region.

ARTICLE II.—THE CARDIAC REGION.

The extent of cardiac percussion dulness will differ according to the force used in percussion, whether slight or great. Gentle percussion detects dulness only where the heart is uncovered by lung ; this is the area of superficial dulness. Stronger percussion detects the dulness of the heart where it lies behind the lung ; this is the area of deep dulness : and it only is indicative of the size of the heart.

i. The Superficial area is roughly triangular in shape, the right side of the triangle being the midsternal line from the level of the fourth chondrosternal articulation downwards, the hypotenuse being a line drawn from the same articulation to a point immediately above the apex-beat, the base being a line drawn from immediately below the apex-beat to the point of meeting between the upper limit of liver dulness and the midsternal line. The area of superficial dulness is much diminished by a deep inspiration, much increased by the patient lying upon the left side (the same position which displaces the impulse to the left), and not much affected by the patient lying upon the right side.

ii. The Deep area reaches upwards as high as the third rib (in children even as high as the second interspace); to the left about a finger's breadth to the left of the impulse; and to the right as far as a little beyond the right margin of the sternum. But, in truth, the right limit of cardiac dulness is not very trustworthy, the osteal and conducted pulmonary notes interfering much with the cardiac percussion-sound.

No doubt it is sometimes quite easy to be able to discover the lower margin of the heart by percussion: sometimes a heightening of pitch and increase of resistence are tolerably well marked on passing from the heart to the liver: sometimes there is a distinct band of faint resonance between the two organs; and sometimes, in passing from the hepatic to the cardiac region, one becomes sensible of a slight increase in the intensity of dulness, and a most distinct increase in resistence, owing probably to comparative thinness of the left lobe of the liver, and its position over the stomach. When the heart and liver dulnesses pass indistinguishably into each other, we must be content with assuming the lower margin of the heart to correspond with a line drawn from a little below the apex-beat of the heart to the point of meeting between the right limit of cardiac dulness and the upper limit of hepatic dulness, bearing in mind how close is the connection between the heart and liver by means of the vena cava.

ARTICLE III.—THE MEDIASTINAL REGION.

The contents of the mediastinum in a healthy state do not affect the percussion-note in any way.

SECTION III.

PERCUSSION OF THE CHEST IN DISEASE.

ARTICLE I.—THE PULMONARY REGION.

AUENBRUGGER'S DICTUM.—If, over the fore-described pulmonary region, we perceive not the fore-described pulmonary sound, equal on both sides, the force of percussion being equal, we may predicate the existence of disease where the sound is unnatural.[1] This is Auenbrugger's dictum, and comprises the whole theory of percussion.

¶ I.—PULMONARY PERCUSSION-SOUND.

The typical pulmonary-note may be departed from by increase or by diminution of its resonance. Hence two classes of unnatural sounds.

Class I.—Increased Resonances.

The resonance is increased by the tone becoming tympanitic (p. 62) or clear.

1stly. Tympanitic Resonance, mostly muffled, seldom or never clear, occurs when the sonorous column of air is greater than natural, a condition present in pneumothorax, and in emphysema of the lung.

2ndly. Clear resonance, sub-tympanitic or tracheal, is afforded by the following conditions :—

i. *a*. Lung which is relaxed as a whole by pleural effusions, or by tumours or enlarged organs without the lung. But, inasmuch as the relaxed lung amid these circumstances cannot keep up its contact with the whole of its chest-wall, the extent of the clear percussion-note is small. *β*. Lung around sharply-defined pulmonary consolidations. *γ*. Lung in which solid or liquid exudations are intimately mingled with air-containing tissue: hence the clear tracheal tones sometimes yielded by catarrh, œdema, congestion of the lung, pneumonia at its very outset or during resolution, phthisis and tubercle.

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[1] "Si igitur ex prædictis locis sonoris non percipitur sonus manifestus, utrique lateri æqualis, eidem percussionis intensitati conformis, morbosum quid in pectore latere significat." Inventum Novum. § 11.

ii. Cavities, filled wholly or mainly with air, when at the surface of the lung, or, if deeply-seated, separated from the surface by dense solid tissue, may afford a clear sub-tympanitic or tracheal tone.

iii. Solid masses, coming to the surface, and closely connected with large air-tubes, sometimes yield a clear tracheal tone: pneumonia and pulmonary tumours may exemplify this fact.

CLASS II.—DIMINISHED RESONANCES.

The resonance is diminished when the tone becomes less loud and long, or more muffled. When absolute tonelessness is present, it is vain to draw (so far as the sound is concerned) any further distinctions. When a certain amount of tone is preserved, it is not only possible but useful to distinguish the degree of clearness and the height of pitch.

The following conditions are attended by diminution of pulmonary resonance :—

i. Extreme distension of the lung with air. Hence we should never percuss a chest whilst the patient is coughing or holding his breath.

ii. Liquid and solid exudations into the lung which do more than relax it. Catarrh, œdema congestion, pneumonia, induration, phthisis, often illustrate this fact ; hæmorrhagic infarctus, cancerous and other tumours more seldom.

iii. Collapse of the lung.

iv. Liquid exudations into the pleura, whereby the lung is deprived of air, and is moreover separated from the chest-wall. But liquid is a good conductor of sound. Were resonant material behind the effusion, percussion would bring forth tone. The dulness shows that there is no resonant material behind the effusion ; in other words, that the lung is deprived of air. That the dulness of a pleural effusion depends upon the state of the underlying lung is proved by these facts. *a*. A considerable pleural effusion may co-exist with a percussion-sound muffled in tone but far from absolutely dull. β. When patients, who are put under the influence of chloroform with a view to paracentesis, struggle and scream, or simply breathe deeply, parts which before were wholly dull to percussion become resonant; yet, puncture these parts, and liquid freely flows. γ. When pleural effusion has been removed by absorption or drainage, the dulness continues to be almost or quite as great as ever, so long as the lung remains unexpanded.

CLASS III.—METALLIC BY-SOUNDS.

I. The Metallic Ring is sometimes heard on percussion of pneumothorax, and of very large pulmonary cavities containing air.

II. The sundry causes of Cracked-Pot sound have been already set forth (p. 73).

¶ II.—PULMONARY PERCUSSION RESISTENCE.

Whatever diminishes the elasticity of the part percussed increases the sense of resistence to percussion. Wherefore massive consolidation of the lung, liquid pleural effusions, and extreme distension of lung or pleura with air, afford a feeling of resistence more or less increased.

¶ III.—EXTENT OF PULMONARY REGION.

Over the cardiac region, percussion is employed in order that we may learn whether the lung has shrunken away from the heart, or covers it, or whether the pleura contains air. In the diagnosis of adherent pleura, of phthisis, and of emphysema of the left lung, these signs become useful.

The discovery of the position of the diaphragm and mediastinum, depends chiefly upon percussion. These subjects will be discussed hereafter.

Article II.—The Cardiac Region.

Disease of the heart itself diminishes or increases the area of cardiac dulness.

i. The area is diminished in the very uncommon case of pneumopericardium, clear resonance superseding the natural dulness. Atrophy of the heart does not much affect the area of dulness.

ii. The area is increased in enlargement of the heart, and in pericardial effusion : for the diagnosis between these conditions the reader is referred to the second part.

Moreover, the area of dulness may be altogether displaced, in consequence of displacement of the heart (p. 41).

ARTICLE III.—THE MEDIASTINA.

Dilatation of the large vessels, aneurysms, and solid tumours in the mediastinum are sometimes the cause of more or less dulness to percussion where there should be resonance; as will be further shown in the second part.

"Pulsa, dignoscere cautus
Quid solidum crepet."

PERSIUS: v. 24.

CHAPTER V.

AUSCULTATION.

SECTION I.

INTRODUCTORY.

ARTICLE I.—HISTORICAL.

AUSCULTATION existed not before Laennec; and "clinical observation, though never blind, had been always deaf." True, a few passages are found in the writings of earlier physicians[1] which speak of sounds heard in the

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[1] The following are the chief notices of auscultation which I have found in authors older than Laennec:—

i. Rales. Hippocrates, Progn. § 14: De Morbis: ii. § 61. Caelius Aurelianus, Acut. Morb. lib. ii. cap. 14.

ii. Leather sound of pleural friction. Hippoc. de Morbis: ii. § 59; ibid. iii. § 16.

iii. Succussion-splash. Hippoc. de Morbis: i. §§ 6, 15; ii. § 47; iii. § 16. De Intern. Affect. § 23. Coacæ Prænot. 424. De Locis in Homine: § 14.

iv. Metallic tinkle. Willis: Phar. rat. part. II. sect. i.: cap. 13. 1674.

v. Pneumonic crepitation. Van Swieten: Comment. in Aphor. 826: 1745.

vi. Respiration bruissante. Double: Séméïologie chest, but the observations remained mere curiosities, wholly without influence upon practical medicine. It is interesting to mark the dawning of the great discovery. Corvisart had studied the different forms of enlargement of the heart, and had endeavoured to distinguish between active and passive enlargement; to this end the character of the impulse was carefully observed. Bayle, a disciple of Corvisart, was in the habit of applying to the heart-region his ear rather than his hand,[1] inasmuch as a heaving impulse is more readily

générale, vol. ii. p. 31 : 1817. See an amusing tale of Double's claim to the discovery of auscultation, in Lond. Med. Gaz. vol. xi. : p. 189.

vii. Heart sounds are briefly alluded to by Forestus (died 1597. Opp. omnia; edit. 1653: vol. ii. p. 241), Harvey (De motu cordis; cap. v.), Stalpart vander Wiel (Observ. rar. i. obs. 36), and others. The clearest account of a murmur is that by James Douglas (Phil. Trans. for 1715 : No. 344 : quoted by Norman Moore, St. Barthol. Hosp. Rep. vol. 26, p. 165). See also Rutherford, as quoted by Allan Burns (Dis. of Heart, 1809 : p. 187 ; a case of mitral disease).

viii. Sounds of fœtal heart. Mayor: quoted by Laennec, ii. p. 459.

[1] "I have found this method nowhere alluded to, and Bayle was the first whom I saw employ it, when we followed the practice of Corvisart together. The professor himself never put his head to the chest." Auscultation médiate, 2me édit., vol. i. p. 5.

detected thereby, be the reason what it may. Laennec, Bayle's friend and fellow-student, adopted the same method. Laennec had undergone a fifteen years' training in the hospitals of Paris, when, in 1816, he was consulted "by a young person who presented the general symptoms of disease of the heart, and in whom palpation and percussion gave no information, on account of the patient's fatness. Her age and sex forbade an examination of the kind just mentioned (by putting the head to the chest), when I remembered a well-known acoustic fact, that if the ear be applied to one end of a plank, it is easy to hear a pin's scratching at the other.[1] I conceived the possibility of employing this property of matter in the present case. I took a quire of paper, I rolled it very tight, and applied one end of the roll to the præcordial region; then leaning my ear on the other end, I was surprised and pleased to hear the beating of the heart much more clearly than if I had applied my ear directly to the chest."[2] He had discovered auscultation. At the Hôpital Necker he explored the new world gained for science; and in 1818 he read a "Memoir upon Auscultation by divers acoustic instruments employed as means of investigation in the diseases of the thoracic viscera, and especially in pulmonary Phthisis." In 1819 he published the first edition of his book on Mediate Auscultation;[1] and, in 1826, the second.

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[1] This remark would seem to imply that Laennec had put his head to the chest, in order "to hear the beating" of the heart: a phrase which he constantly uses: compare Auscultation médiate: vol. ii. pp. 385 sqq.

[2] Auscultation médiate: vol. i. p. 7. Montaigne's (Essays, ii. chap. 37) remark that "the physicians have

ARTICLE II.—METHODS OF AUSCULTATION.

It was by means of an instrument[2] that Laennec was enabled to discover the powers of Auscultation. Since his day the stethoscope has been discarded by many persons, who, preferring immediate to mediate auscultation, apply the ear directly to the chest. And doubtless

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no speculum matricis by which to discover our brains, lungs, and liver," has ceased to be true with respect to the lungs: the stethoscope is our speculum pectoris.

[1] René Théophile Hyacinthe Laennec: De l'Auscultation médiate ou traité du diagnostic des maladies des poumons et du cœur, fondé principalement sur ce nouveau moyen d'exploration. Paris, 1819. For title of second edition, see p. 51.

[2] One of those instances of the door or gate (in Bacon's phrase: Nov. Org. ii. chap. 39) "which assist the operation of the senses."

the sounds are heard loudest in this way; they are weakened by conduction through a stethoscope. However, the drawbacks to immediate auscultation are great, and chiefly these: the impossibility of listening to every patient's chest without the interposition of some kind of vestment, which, besides being a bad conductor of sound, gives rise to divers noises of its own; the impossibility of applying the ear to every portion of the chest; the impossibility of localising sounds with sufficient accuracy. Moreover, we must not take for granted that the ear, applied directly to the chest, will needs conduct the sounds more truly than will a stethoscope. Heart-sounds and murmurs, in particular, often acquire by immediate auscultation, a booming, indistinct character which does not belong to them. When this is the case, what we lose in mere loudness by using the stethoscope is well repaid by the greater definiteness of what we hear. No doubt a person may become a skilful auscultator who listens with his unassisted ear; but let mediate auscultation ever be considered the rule of practice, and immediate the exception; the physician making of the stethoscope, not a crutch, but a staff, which he uses when he has it, yet when he has it not he does not want it.

Stethoscopes are solid (rods) or tubular (pipes); tubular stethoscopes are rigid or flexible; flexible stethoscopes are single or binaural; so that there are four kinds of stethoscopes in use. Solid stethoscopes are made of wood; rigid tubular stethoscopes of wood, gun-metal or vulcanite; flexible stethoscopes of caoutchouc. A flexible stethoscope conducts wholly by the column of air contained within the tube; when the pipe is plugged, it conducts no longer. Scarcely need it be said that rigid pipes conduct partly by the bore and partly by the tube; and that solid rods conduct wholly by the solid.

To compare the rigid with the binaural stethoscope would require more time and study than the subject seems to deserve. It is a mistake to assume that we can auscultate best with the binaural flexible stethoscope which enables us to listen with both ears. Mere loudness is the least important property of breathing and vocal sounds, and in my opinion, a rigid tubular instrument often conveys the other properties of sound more truly, and thus leads to more refined auscultation. But a man rightly prefers that kind of stethoscope to which he has been most accustomed.

Article III.—Murmurs in General.

A murmur is a sound produced by the flow of fluid, liquid or gaseous, along a tube. Fluid flowing, however swiftly, along a tube of equal caliber throughout, produces no sound.

I. Onward Murmur.—A jet of fluid flowing swiftly enough, out of a narrow orifice into a wider space, is called a fluid vein.[1] The molecules of a fluid vein are agitated by movements which cause the vein to vibrate, and which are productive of sound. The loudness of the sound depends upon the swiftness of the flow: the quality of the fluid, and the size of the orifice are of import only inasmuch as they exert an influence upon the swiftness of the flow. The sound is carried farthest in the direction of the flow, hence the name onward murmur.

II. Backward Murmur. — Fluid flowing, however swiftly, from a wider into a narrower space, produces no sound. But if the stream break, swiftly enough, upon a bevelled edge, like unto that of a whistle, the fluid becomes vibrating and sonorous. If the bevelled edge surround a considerable constriction within the tube, the sound is carried farthest in a direction against the flow of fluid: hence the name, backward murmur.[1]

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[1] Félix Savart: Annales de chimie et de physique: 2nd series, vol. 53, p. 337. Paris, 1833.

III. MUSICAL MURMURS.—Murmurs of either kind sometimes acquire the character of tone; these are called musical murmurs.

i. A musical murmur is usually due to the consonance of a solid, which plays the part of the reed in an instrument, and which is set in vibration by the sonorous fluid stream.

ii. A musical murmur is sometimes due to vibration of the fluid vein alone; especially when the tube is curved so as to form a semicircle just beyond the constriction which causes the fluid vein.

SECTION II.

AUSCULTATION OF THE LUNGS AND PLEURÆ.

Auscultation of the surface of the chest is practised with reference to three kinds of sounds: i. the Voice, as it is heard over the thorax: ii. the sounds of Breathing: and iii. sounds produced in the Pleura.

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[1] Bergeon: Des causes et du mécanisme du bruit de souffle, p. 25. Paris, 1868.

ARTICLE I.—THORACIC VOCAL SOUNDS.

The voice, as it is heard by auscultation, is usually called Vocal Resonance: a phrase which we inherit from Laennec, and which involves a theory not yet discussed. It is useful to study vocal resonance before the respiratory sounds; inasmuch as the origin of the sound is, in the former case, clear and indisputable, but, in the latter case, not so. Let us follow the historical order: the first fact which Laennec discovered by auscultation concerned vocal resonance.

¶ I. THEORY OF VOCAL RESONANCE.

The prime distinction between the different kinds of vocal resonance lies in the degree of change which the tone of the voice has undergone by the time it reaches the surface of the chest. Whence two classes of vocal resonances; the muffled (or more changed), and the clear (or less changed). Clear vocal resonance, being usually heard over the large air-tubes, is also called bronchophony.

The term, clear, as applied to vocal resonance, expresses a complex notion. Using words which will be explained a few pages onwards, vocal resonance may be called clear in respect either of the fundamental tone of the voice, or of the articulated over-tones. The fundamental tone is clear in proportion as it retains its musical laryngeal character; and the articulated over-tones are clear just in proportion as they are distinct; after transmission through the lung.

i. MUFFLED VOCAL RESONANCE.—The ear, applied to the chest of a person who is speaking, perceives a humming or buzzing sound: the laryngeal tones have lost their clearness, and distinctness: their loudness also is much diminished: so that a weak voice is not heard at all.

ii. CLEAR VOCAL RESONANCE, OR BRONCHOPHONY.—What vocal resonances shall be deemed clear enough to form a class apart from muffled vocal resonances is an arbitrary arrangement. The line is drawn at that amount of clearness which the voice possesses in a certain proportion of perfectly healthy persons, when listened to in the upper part of the interscapular regions. There, in some people, we hear nothing different from what we hear over the rest of the chest; but in some we hear a vocal resonance which is comparatively clear, that is to say, which approaches more nearly in character to the sounds heard over the larynx or pharynx. Bronchophony, then, I define to include the clearest vocal resonance ever heard over the healthy chest, and all degrees of clearness greater than this. For disease sometimes affords thoracic vocal sounds which are far clearer than any heard in health ; sounds which are not more muffled than those heard over the larynx. And, just as the least clear vocal resonance, which can be called bronchophonic, is that which is heard over the healthy bronchi ; so does auscultation of the larynx afford a standard of the most clear vocal resonance. For it is very seldom possible that the voice should be heard over the chest, in health or in disease, with greater clearness than over the larynx.

The clearness of vocal resonance is not dependent upon its loudness ; weak sounds may be as strongly bronchophonic as the loudest. Indeed, with respect to articulated sounds, the two characters of clearness and loudness are often opposed, so that the bronchophonic resonance is rendered indistinct by its loudness ; the ground-tone of the voice drowns the articulated over-tones. When this is the case, we must seek to set aside the element of loudness; a consideration which leads us on to the topic of

Whispered Bronchophony. Now whispering is articulation of the breathing sounds, the glottis being passive. And the bronchophony f whispering is often clearer than that of the ice. Sometimes bronchophony cannot be tected at all unless the patient whisper.

ectoriloquy. The name given by Laennec hat physical sign which was the subject of arliest publication relative to auscultation. riloquy, having been the first-fruits of his ery, no wonder that he always clung with n to the name and sign. Laennec meant riloquy, a very clear vocal resonance o characters; first, the being conveyed, along the bore of the stethoscope to e observer: next, the being heard small space of the chest. Loudness of the vocal resonance has nothing to do with pectoriloquy; a whisper may possess the two notes of apparent transmission and exact circumscription. But the distinction drawn by Laennec between pectoriloquy and bronchophony is unsatisfactory. The desire of making pectoriloquy to be a sign pathognomonic of accidental cavity within the lung led Laennec to speak of perfect, imperfect, and doubtful pectoriloquy : now he himself confesses that he could not distinguish doubtful pectoriloquy from bronchophony. In short, he wavered in his own definition ; a fact known to his pupils.[1] If we are to retain the word pectoriloquy in use, it can only be by taking it to signify strictly breast-speech, that is to say, bronchophony which is especially clear and distinct in respect of the articulated overtones.

Ægophony. Another kind of bronchophony is that which Laennec called ægophony, in order to indicate its likeness to the bleating of a goat : still closer is the comparison which he makes between ægophony and the voice of Punch. Laennec's idea of ægophony, like that of pectoriloquy, was confused by his desire to make the sign pathognomonic; he thought it indicative of pleural effusion. His own pupils could not follow him :[2] nor will we ; for ægophony is nothing but bronchophony with a nasal quality more marked than usual.

Vocal resonances are mostly heard best by auscultation with the ear alone; sometimes, indeed, the stethoscope altogether fails to conduct them. The same is true of auscultation of a pitch-pipe's sounds.

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[1] Harrison : Lond. Med. Gaz. 1836 : vol. xix. p. 457.

[2] Piorry : Percussion médiate, p. 84.

Vocal resonance, though useful chiefly as a confirmation of the notions acquired by auscultation of the respiratory sound, sometimes possesses an independent value. When a patient breathes so as to produce scarcely any sound, or breathes noisily, or otherwise unnaturally, then we call in the aid of vocal resonance to remove our difficulties. A kind of artificial vocal resonance is produced by causing the patient to sound a pitch-pipe during auscultation.

¶ II.—PHYSICAL CONDITIONS OF VOCAL RESONANCE.

Vocal resonance has been defined to signify the voice as it is heard upon the surface of the chest. Wherefore two points demand examination : What is the voice ? How does it reach the surface of the chest ?

The larynx is a reed instrument which is capable of sounding a fundamental tone. In speaking, sundry over-tones are produced along with the chief tone. The vocal chords may vary the pitch of the tone they produce, but no change in the quality (timbre, clangtint) of the sound can be effected in the larynx itself. That is to say, the larynx cannot articulate. The mouth, however, by varying its shape, can be made to resound to different laryngeal tones; and it is to different associations of these tones, occurring in the mouth, that articulation is due.[1]

The next consideration concerns the manner in which the voice reaches the surface of the chest. The sonorous undulations generated within the glottis and articulated above it, pass upwards and downwards; and just as the pharyngeal vault, the nasal fossæ, and the mouth play the part of an arched roof, so does the trachea that of a speaking-trumpet. The inner surface of the windpipe reflects the vocal vibrations which would otherwise diverge and confines them in the tube, so that the voice is carried in all its fulness down the windpipe; and would be heard at the bifurcation of the trachea, as well as in the mouth, were it not for two circumstances, namely, that the current of air is in a direction reverse to the propagation of the sound, and that the articulated tones have to pass through the narrow glottis. Hence two causes of change in the thoracic voice to begin with.

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[1] Tyndall: Sound, pp. 197 seq. See a curious anticipation of this doctrine in Shaftesbury's Characteristicks: Soliloquy, part 3, sect. 1: first published in 1710.

But the analogy of the speaking-trumpet ceases at the bifurcation of the trachea. Clear vocal resonance is not heard, in healthy people, beyond the neighbourhood of the larger bronchi: natural bronchophony ceases there. We have now to consider the physical conditions of the lungs. First of all we must remember that they are kept in a state of permanent openness or distension which favours the conduction of sound along the air-columns within the tubes. But, on the other hand, the progressively increasing number of air-tubes renders the sound they conduct to any given spot progressively weaker: the voice is no longer confined within a single cylindrical tube, but is spread out and diffused by an enormous number of minute diverging tubes, having a total sectional area very much greater than that of the single tube whence they spring. Probably the diminishing rigidity of the walls of the bronchia diminishes their reflective power: probably the increasing surface of tissue exposed to the sonorous vibrations increases the conduction of those vibrations, by the tissues away from the air-columns. Wherefore the voice, heard over the surface of the lungs, has lost both in loudness and clearness, is both weak and muffled. But why is the bronchophony of the larger tubes not heard above the vesicular vocal resonances? Because pulmonary tissue in its naturally distended state, consisting of incessant alternation of air and membranous walls, is a bad conductor of sound, whether as to loudness or to clearness of tone; and the bronchophony must pass straight through the lung.

ACCIDENTAL BRONCHOPHONY AND PECTORILOQUY.—Accidental bronchophony is the name given by Laennec[1] to bronchophony which is heard at a spot where nothing beyond muffled vocal resonance would be heard in a healthy chest. Accidental bronchophony is due to increased conducting or reflecting power in the lung tissues.

i. Increased Conduction.—Vocal resonance, heard at the surface of the lung, is muffled in consequence of having to pass through badly conducting material, namely, the lung. Now the conducting power is increased by whatever increases the homogeneity of the structure of the lung, or, in other words, by whatever makes the lung approach nearer to simple solid or to simple air. The former case only, that of solidification, will be discussed in this place; the latter case of aërifaction, is complicated by conditions of reflection. Solidification includes collapse of the lung, exudations into it, and new growths. Simple collapse brings the larger bronchia nearer to the surface, and so promotes bronchophony. Such is not the case in exudative consolidations, yet they also increase the lung's conducting power. The note of a pitch-pipe, sounded between the lips, is heard more clearly over pneumonic lung than over healthy lung. A solid nodule, close to a large bronchium on the one side, and reaching the surface of the lung, has been known to conduct (perhaps to magnify) the bronchial voice so well as to render it deserving of the name of pectoriloquy.[1] A coincident pleural effusion brings in a new element of heterogeneity. The sonorous columns of air in the bronchia have to give up their vibrations to the condensed lung, which yields them in turn to the fluid contained in the pleural sac; from the fluid effusion they have to pass through the solid walls of the chest. Yet liquid is a good conductor of sound: over a serous pleural effusion we may sometimes hear accidental bronchophony so clear that we can distinguish any number the patient speaks. Nevertheless the bronchophony often becomes weakened and muffled. Moreover, it sometimes becomes changed in quality by passage through the pleural fluid, that is to say, it becomes ægophonic or amphoric, according as the fluid is liquid or aërial.

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[1] Auscultation médiate, vol. i. p. 65.

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[1] Walshe: Diseases of lungs, 3rd edit. p. 147.

ii. Increased Reflection.—The voice is conveyed along and within the air tubes, by constant reflection of its sound-waves. In the spongy structures reflection ceases, so far as the human ear is concerned. But if they be replaced by a cavity, not beneath the bronchia in reflecting power, bronchophony will take the place of a muffled vocal resonance. If the cavity be large, and its inner surface apt for reflection, the bronchophony (" reverbs a hollowness ") acquires a prolonged hollow and reverberating character.

Accidental Bronchophony is oftentimes clearer than the natural bronchophony heard over the upper dorsal vertebræ, sometimes clearer than the tracheophony heard just above the sternum, very seldom clearer than the laryngophony heard over the larynx, never clearer than the voice from the mouth.

Ægophony.—i. Laennec believed that the smaller bronchia, especially those with walls bereft of cartilage, become flattened by a pleural effusion, so as to behave like the reed of a bassoon or hautboy. Thus the bronchial tree becomes a sort of wind instrument terminated by a multitude of reeds in which the vocal resonance quivers.[1] ii. Wintrich believed that the ordinary nasal character in bronchophony is due to a strong vibration of the walls of bronchia so small that it is accompanied by actual collision of their opposed internal surfaces. The vibrating column of air, broken incessantly, yet too rapidly for the intervals to be distinguished by the ear, imparts a nasal sound. When the interruptions to the sound become sensible to the ear, they yield the bleating character.[2] iii. Stone believed that it is not the pure laryngeal fundamental tone of the voice which affords ægophony, but only the articulated overtones, whether whispered or spoken aloud. A layer of fluid in the pleura, while it stops the larger and coarser vibrations of the groundtones, lets pass the finer and closer undulations of the high harmonics.[1] iv. For my own part I believe that ægophony is no more than a high degree of the nasal quality of bronchophony; and that nasal bronchophony is really a rhinophony,[2] which acquires its nasal character by resonance of the voice within the pharyngeal vault.

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[1] Laennec: Auscultation médiate, vol. i. p. 79.

[2] Wintrich: Einleitung, pp. 119, 146 seq.

To conclude: excepting a few uncommon cases (p. 106 § ii.) it seems that resonance (in the strict acoustic sense) has nothing to do with the production of vocal resonances in health or disease; that in fact the phrase, vocal resonance, can be continued in use only with the distinct understanding that the word resonance is employed in a sense quite peculiar to the nomenclature of auscultation.

¶ III.—VOCAL RESONANCE IN HEALTH.

Vocal resonance over the pulmonary regions is weak and muffled. Behind, opposite the trachea and the bronchial bifurcation (over the upper dorsal spines), bronchophony is heard in many healthy persons : a circumstance probably accounted for by the fact that only solids intervene between the windpipe and the surface of the body at the spot indicated. Under the clavicles, near the sternum, the vocal resonance (especially on the right side, and in women) is often bronchophonic. The voice, in women and children, often cannot be heard over a large part of the chest.

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[1] W. H. Stone: St. Thomas' Hosp. Reps. 1871, vol. ii. p. 187.

[2] A term of Laennec's: Ausc. méd. i. p. 126.

¶ IV.—VOCAL RESONANCE IN DISEASE.

1. Increased Clearness of Resonance : Accidental Bronchophony.—i. Whatever increases the conducting power of the spongy structure of lung increases the clearness of vocal resonance. It has been already shown that the conducting power of lung is increased by whatever diminishes the heterogeneity of its structure. Diminution of the quantity of air contained in a part of the lung, diminution of the number of alveolar septa ; either of these changes will be attended by increased homogeneity. For these reasons it will be apparent why consolidation of lung and cavity are the two causes of accidental bronchophony. Consolidation of lung includes simple collapse, hæmorrhagic infarctus, pneumonia, cirrhosis, phthisis, tubercle, and cancer. Cavities are due

to phthisis, dilatation of bronchi, gangrene, or great emphysema.

ii. Cavity, however, when its inner surface is apt for reflection, introduces a new element into the case. But let it be re-iterated that, in practice, the bronchophony of cavity cannot be distinguished, with absolute certainty, from that of consolidation.

iii. Collapse of lung, attended by bronchophony, will be extensive enough to include large open air-tubes. And this is seldom the case, unless the collapse be due to pleural effusion, liquid or aërial. In the latter case, that of pneumothorax, vocal resonance, whether bronchophonic or not, becomes amphoric.

2. Diminished Clearness of Resonance is an unimportant sign, except it be known to have supervened upon resonance unnaturally clear. And, in such cases, the soniferous power of the bronchi is at fault, in consequence of collapse, or of obstruction by mucus, blood, or exudation.

Resonance of Cough and Cry. In infants the thoracic resonance of the cry sometimes affords useful evidence of disease ; for instance, the cry heard through hepatised lung is strongly bronchophonic. In like manner, the resonance of cough sometimes acquires value when other signs are absent. Much of what has been said of the voice applies to the cough and cry.

ARTICLE II.—RESPIRATORY SOUNDS.

¶ I.—THEORY OF RESPIRATORY SOUNDS.

The fundamental division of respiratory sounds is the same as that of vocal resonances. The respiratory sound, heard over the larger air-tubes, differs, by the possession of a certain quality, from that heard over the spongy structure of lung. Wherefore the former is called Bronchial, and the latter Vesicular breathing.

i. VESICULAR BREATHING.[1]—The ear, applied to the breathing chest, detects a sound which may be defined by the negative property of not possessing the bronchial quality. Wherever breathing lung is in contact with the chest-wall, there we hear this sound. Its inspiratory portion has a duration equal to that of the inspiratory movement; the expiratory portion follows after the shortest possible interval, has a duration only one-fourth or one-fifth of that of the inspiratory sound, and is much less loud than it.

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[1] Term suggested by Mériadec Laennec to replace the "bruit respiratoire pulmonaire" of René Laennec. Notes et additions, &c. Paris, 1836 : p. 18.

The sound is loud in proportion to the rapidity and depth of the breathing. The louder the sound the greater becomes the relative duration of the expiratory portion : but mere prolongation of expiratory sound is a sign of no importance. Sometimes the tranquil breathing of adults is unattended by expiratory sound. Sometimes, especially in fat middle-aged women, the breath sound can be hardly heard at all. Loud vesicular breathing was called Puerile by Laennec.[1]

Sometimes the inspiratory sound, instead of being continuous, is divided into three or four distinct parts ; sometimes the expiratory sound likewise is duplicate : this is called jerking breathing : a sign of uncertain cause and no importance.

ii. BRONCHIAL BREATHING. — Bronchial breathing, like bronchophony, is heard about the seventh cervical, and three or four upper dorsal vertebræ in many healthy persons : especially in those who are thin, in women, and in children.[2] Bronchial breathing is distinguished from vesicular breathing by the possession of a special quality of sound, which is best called hollow or reverberating.[1]

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[1] Auscultation médiate, vol. i. pp. 49 et sqq.

[2] Laennec : Auscultation médiate, vol. i. pp. 55, 56.

The loudness of bronchial breathing is an unimportant property: the special quality is as well marked in weak respiratory sounds as in the loudest; nay, loudness of sound often proves an impediment to the detection of that quality which alone constitutes the note of bronchial breathing. The expiratory sound, although usually less loud than the inspiratory, often manifests the special quality better. Not that this is always the case, sometimes (but not often) the bronchial quality is possessed by the inspiratory sound in a higher degree than by the expiratory. Sometimes (but seldom) the expiration of bronchial breathing is wholly inaudible, the inspiratory sound being, at the same time, highly bronchial: a proof, were proof needed, that prolonged expiration is no essential property of bronchial breathing.

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[1] Laennec: Auscultation médiate, vol. i. p. 55. But as P. M. Latham says, "The sounds can only be learnt by the practice of listening to them. It is useless to describe them. They are simple perceptions of sense, which no words can make plainer than they are, when the ear has once become familiar with them. I must leave you to be your own self-instructors, and recommend you to be constantly practising auscultation for the purpose." Diseases of the Heart: Lecture I.

Bronchial breathing sometimes loses its peculiar quality by conduction through stethoscopes of a certain make: and an instrument with a bore too large is especially likely to bring about this unfortunate result. In such a case immediate auscultation must be practised.

Laennec[1] gave the name Cavernous to that kind of breathing which possesses the bronchial or hollow quality in a high degree: not that cavernous breathing always indicates cavity.

Another kind of bronchial respiration, which has been named Tubular,[2] is characterised by a well-marked whiffing quality. It is most frequently heard over hepatised lung.

By the name of souffle or puff, Laennec[3] designated a phænomenon sometimes superadded to bronchial, cavernous, or tubular breathing. The air during inspiration seems to be drawn away from the ear of the observer, and during expiration to be puffed back again: a sign of no importance.

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[1] Auscultation médiate, vol. i. p. 57.

[2] The word tubular was first used by Laennec, with respect to the cough. Auscultation médiate, vol. i. p. 90.

[3] Auscultation médiate, vol. i. pp. 58, 421, 423.

By the name of souffle voilé, or veiled puff, Laennec[1] designated a phænomenon which it has puzzled his successors to identify. It is a modification of the puff, he says, in which each vibration of voice, cough, or breathing seems to shake a sort of mobile veil placed between a lung cavity and the observer's ear. Skoda[2] believed that Laennec referred to the condition of an inspiratory breathing sound, indistinct at its commencement, and suddenly becoming bronchial. Skoda's veiled puff is not very uncommon.[3]

¶ II.—PHYSICAL CONDITIONS OF RESPIRATORY SOUNDS.

The physical conditions of vocal resonance, as already set forth, and those of the breathing sounds are very much alike. The main question, which we have now to treat, concerns the source of the breathing sounds ; that is to say, the manner in which they are produced. I may as well at once declare that, for my own part, I hold to Beau's doctrine in this matter.[1] Let the reader call to mind the fact that whispered sounds (in other words, articulated sounds produced by respiration in an inactive glottis) are audible all over the chest, and he will then be more ready to agree that it is at least possible, not to say probable, that ordinary unwhispered respiratory sounds are produced in the same place. The lungs in the chest are distended : the tubes are all open, apt for conduction and reflection. The passage of breath through the narrow glottis into wider spaces above and below, produces sonorous fluid veins inspiratory and expiratory. This glottidean breathing murmur, modified by resonance in the pharyngeal vault above and the windpipe below, is conducted down the air tubes just as the loud or whispered voice is carried. And all that has been said concerning the conduction and reflection of vocal resonance applies to the respiratory sounds.

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[1] Auscultation médiate, pp. 59, 213, 421, 423, 449.

[2] Markham's translation, p. 99.

[3] All reference to some terms in frequent use (such as harsh, coarse, rough) applied to breathing sound, has been eschewed, because their meaning is ill-defined and not correspondent with clear and distinct ideas. Even supposing that any such word denote a definite quality of sound, unless it signify some certain and constant condition of lung, to recognise the sound is useless. But when we read about "la rudesse de l'inspiration, qui devient râpeuse, granuleuse, au lieu d'être légère, moelleuse, et caressante à l'oreille," we seem to have passed beyond common sense into mere fancy and fine writing.

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[1] Beau : Archives gén. de Méd. Aug. 1834.

It is convenient to look upon the glottis as the only source of the breath-sound.[1] But, applying the general principles of murmur-production (p. 94) to the air-passages, we must admit that the nose, mouth, throat, any unevenness of the windpipes, the bifurcation of tubes, and the mouths of air-sacs, must all be deemed possible causes of onward or backward murmurs.

Bronchial breathing is the glottidean sound as heard in the larger bronchia. Vesicular breathing may be supposed to lose the bronchial hollow quality in consequence of a badly conducting material (the spongy texture of lung) through which the sounds have to pass. Puerile breathing implies louder glottic sounds than usual and a very open state of lung. Cavernous breathing is bronchial breathing rendered more intense by the reverberation of a cavity. Tubular breathing is hard to explain ; we do not know how it acquires its peculiar whiffing character.[1] The puff probably shows that the seat of the bronchial breathing is near the surface. Skoda's veiled puff is most likely due to sudden removal of an obstruction in the tube, which communicates with a cavity.

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[1] "The facts are not easy of explanation. Reasoning resembles the universal solvent of the alchemist; if we believe nothing but what will withstand the attack of our logic, we shall believe very little. Yet it is desirable, in spite of reasoning, to hold some opinion concerning the source of the breathing sounds, and as the result of long experience in teaching, I can say that the simple glottic theory is sufficient for all practical purposes. Whether the theory be true is another thing, but 'tis so like truth, 'twill serve our turn as well." Theory of the breathing sounds heard by auscultation: St. Bart. Hosp. Reports, vol. xxvi. p. 105.

¶ III.—RESPIRATORY SOUNDS IN HEALTH.

The breath-sound is vesicular over the whole of the pulmonary regions : excepting that bronchial breathing is to be heard between the scapulæ in many persons ; sometimes under the right clavicle also (especially in women) ; and sometimes, yet seldom, under the left. The loudness of breathing sounds differs very much in different people. In children, the respiratory sound is loud or puerile ; a fact to be explained by the frequency of their respiration, and by the thinness of their chest walls.

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[1] Perhaps the following hypothesis meets the case. When disease consolidates the air-vesicles and bronchiola proceeding from a larger air-tube, and so converts it into a cavity with unbroken walls, it becomes capable of resonance. A current of air, passing across the mouth of such a tube, produces a flutter there, and a certain pulse of this flutter is taken up by the resounding cavity, and raised into a definite sound. An effect concurrent with both inspiration and expiration.

Crepitation due to collapse is sometimes heard, especially at the bases of the lungs, and at the beginning of an examination ; a few deep breaths remove this rale, when it is not due to disease.

¶ IV.—RESPIRATORY SOUNDS IN DISEASE.

I. Weak respiration indicates weak production or conduction of the glottic murmur. Whatever obstructs the air passages within, whatever compresses them without, whatever interferes with movement of the chest, will produce weak respiration ; which, in other respects, may be vesicular or may be bronchial.

II. Loud respiration indicates good production and conduction of the glottic murmur. When local and a consequence of disease, the breathing is often obstructed in some part of the lungs other than that where the loud respiration is heard.

III. Bronchial respiration, heard where naturally the breathing is vesicular, indicates obliteration of the damping spongy structure of the lung: obliteration which may be brought about in two ways:—

i. By collapse of air-sacs, or by exudation into them, or by both processes combined. The pulmonary substance, thus rendered more homogeneous, is better fitted to convey the bronchial sounds to the surface. Pneumonic, phthisical, tubercular, hæmorrhagic, cancerous consolidation; collapse, simple, congestive, or œdematous; and cirrhosis of lung; all produce this effect.

ii. By destruction of air-sacs: whereby the vesicular structure is replaced by cavities, capable of reverberation or not. Phthisis, and dilatation of bronchi, are the most frequent lesions belonging to this class.

Wherefore, in short, consolidations and cavities are the two causes of bronchial breathing, as also of bronchophony.

iii. The case of consolidation, whether mere collapse or other, is sometimes complicated by a concurrent pleural effusion. Under these circumstances, somewhat depends upon the kind of the effusion, but more upon the state of the lung. The sounds in the solid lung are either weak or bronchial, as before explained. A liquid effusion simply conducts the breath sounds, thereby weakening them more or less. An aërial effusion either simply weakens the sounds, or imparts to them an amphoric hum.

¶ V.—RALES.

The natural breath-sound having been described, and also the changes in that sound which are due to disease, we now come to Rales, or sounds which are produced within the lung by respiration, and which are wholly additional to the natural or morbid breath sound. Rale,[1] rhonchus, rattle ; these words are synonymous. I shall retain the classification adopted by Laennec, and also his nomenclature, because I believe that his distinctions are of practical value in diagnosis, and that his names are as good as any others.

Rales are of three kinds: crepitant, mucous, and sonorosibilant.

I. CREPITANT RALE.—A rale which has been well compared to the sound produced by rubbing a lock of the hair between the fingers close to the ear.[2] Crepitation of this kind is heard during inspiration, sometimes throughout the whole of it, sometimes towards the end of it only; sometimes, but seldom, crepitation is expiratory also. Pneumonia, collapse, and œdema of the lung, are the three conditions which afford crepitant rale. Its occurrence in collapsed lung, during a deep-drawn breath, explains the condition necessary to the production of inspiratory crepitation in most cases; namely, the opening up of collapsed air-sacs.[1] Disseminated collapse of single air-vesicles is an important part of the changes consequent upon pneumonia and pulmonary œdema, as any one who will inflate the uncut engorged or œdematous lung of a child may easily discover. Inflation of the lung in such cases will bring out an immense number of air-sacs, which were before invisible because collapsed, and which collapse again directly the air is allowed to escape: the transparent, non-pigmented tissues of a child are particularly favourable for this experiment. In pneumonia so soon as hepatisation becomes dense the collapse cannot be removed by any pressure of air. Sometimes, doubtless, crepitation is a very fine mucous rale. Indeed, the main distinction of crepitant from mucous rale, so far as concerns the sounds alone, lies in the shortness or smallness of each crepitus, and, what is mostly associated herewith, the large number of crepitations attending each inspiration.[1]

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[1] A French Teutonic word : essentially the same as the English word, rattle.

[2] Williams : Lond. Med. Gaz. vol. xxi. p. 275 ; vol. xxii. p. 261.

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[1] Van Swieten: Comm. in Aphor. § 826. "Ingratus in pectore strepitus, qui fit a vesiculis pulmonum siccis hincque crepitantibus instar corii arefacti, dum inspirando extenduntur," vol. ii. p. 724. Lugd. Batav., 1745.

II. Mucous Rale.—This rale includes all sounds which seem to be due to the passage of air through mucus or other fluids contained in the air passages. The notion received is certainly, for the most part, that of bubbles bursting; sometimes the sound is crackling rather than bubbling in character. Mucous rale is sub-divided into varieties, according to the following considerations :—

i. The apparent size of the bubbles; so small as to approach the crepitant rale (subcrepitant), so large as to deserve the name of gurgling, and and all intermediate sizes.

ii. The clearness of the rale: the rale being sometimes more or less obscure,[2] on account of weakness of the respiration; a deeper breath

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[1] After the example set by P. M. Latham and Watson, the term crepitation is still used by many, so as to include mucous rale.

[2] Râle obscur: Laennec: Ausc. méd., vol. i. p. 103.

(when this is possible) bringing out a rale much more distinct.

iii. The reverberation of the rale; when this character is well marked, the rale is called cavernous.[1] Reverberating mucous rale indicates the same physical conditions as do reverberating (cavernous) breathing or voice.

III. SONOROUS AND SIBILANT RALES.— These are rales which are more or less accurately described by such words as snoring, cooing, whistling; low-pitched sounds being called sonorous (rhonchus), and high-pitched, sibilant (sibilus). Sounds of this kind are due to local narrowing of the air-passages; most commonly by mucus, in which case a cough, when it dislodges the mucus, removes the rale. Palpable vibration often concurs with sonorous rale.

IV. DOUBTFUL RALES. — The respiratory sound is sometimes attended by sounds not comprehended in any species of rale hitherto described, and doubtful both as to situation and significance. I allude particularly to two kinds of sounds. First: sundry creaking sounds, not seldom heard at the apices of the lungs, and possibly due to creaking of pleural adhesions, but possibly also produced in the tissue of the lung itself.[1] Secondly: the dry crepitant rale with great bubbles, as Laennec[2] named a sound resembling that produced by inflating a dried bladder, and due, he supposed, to distension of the enlarged air-sacs of emphysematous lung.

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[1] Laennec: Auscultation médiate, vol. i. p. 99.

ARTICLE III.—PLEURAL SOUNDS.

The pleuræ in a state of health yield no sound.

In diseases of the pleura sundry sounds are met with, which may be arranged in two classes; friction sounds, and sounds produced in large cavities.

¶ I.—FRICTION SOUNDS.

Any unevenness of opposed pleural surfaces, and any solid exudation or morbid tissue between them, tend to produce friction sound during respiratory movement.

The discovery of friction sounds has been the most important addition to the practice of auscultation of the lungs, as it was left us by Laennec.[1] Not that he was altogether ignorant of these sounds. In the spring of 1824, Honoré, who had succeeded Laennec at the Hôpital Necker, having observed a peculiar sound in a patient's chest, sent him to Laennec; who heard what he called "up and down rubbing," and considered it to be due to interlobular emphysema.[2] The real meaning of pleural friction was first made out by Reynaud.[3] The diagnosis of friction sound depends upon consideration of the following particulars, but chiefly upon the first:—

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[1] Bruit ou râle de froissement pulmonaire. Fournet: Recherches cliniques, vol. i. p. 172. Paris, 1839.

[2] Auscultation médiate, vol. i. pp. 106, 308, 343.
Louis: Méms. de la soc. méd. d'observn. vol. i. p. 215.

1st. Character of the sound. This is sometimes peculiar; giving a distinct notion, either of rubbing in any degree, between lightest grazing and harshest scraping: or of creaking comparable to the creaking of leather.

2nd. Position of the sound. Friction sound is commonly heard over a small part of one side of the chest; especially its lower part below the axilla, or about the angle of the scapula; here movement of the pleural surfaces upon each other is freest. It is uncommon to hear friction sound on both sides of the chest at the same time.

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[1] I have elsewhere (p. 88) alluded to the fact that Hippocrates knew pleural friction-sounds.

[2] Auscultation médiate, vol. i. p. 115.

[3] Journal hebdomadaire de médicine, 1829, vol. v. p. 563.

3rd. Time of the sound. Friction sound may accompany inspiration, or expiration, or both, or only a small portion of either, especially the very end of inspiration. Friction sound mostly attends the breathing movement only; but sometimes pleural friction, over the cardiac region, attends the heart's movements only, so as to resemble pericardial friction.

4th. Friction sound is seldom accompanied by any unnatural quality of respiratory or vocal sound.

5th. Cough has no power of modifying or removing friction sounds.

The commonest cause of pleural friction is exuded lymph; organised false membranes, miliary tubercles, and mere extra-vascularity, are less common causes. Whether Laennec was right in believing that friction sound may be produced by interlobular emphysema (p. 126) is doubtful.[1]

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[1] Walshe (Diseases of the Lungs, p. 133) and Gairdner (Clinical Medicine, p. 438) agree with Laennec.

¶ II.— AMPHORIC SOUNDS:

Or, sounds produced in a large cavity, are four in number: two spontaneous, and two produced by the physician during examination: the former are amphoric hum, and metallic tinkling; the latter are the bell sound, and succussion splash. I describe these signs in this place because they are most commonly produced in the pleural cavity.

I. AMPHORIC HUM.—By Amphoric hum[1] is meant metallic resonance, such as is produced by blowing, speaking, or coughing, into a large and empty bottle. Amphoric hum requires the existence of a large cavity containing air; and may accompany—

i. The respiratory sounds; when loud bronchial respiratory sounds pass from unperforated lung through a large resounding cavity, and so acquire amphoric quality: or when there is a wide fistulous opening between a pneumothorax and a large bronchus, so that the breath flows freely in and out, a very uncommon condition. Communication between the bronchi and the pneumothorax is certainly not necessary. Breath-sounds seldom become amphoric within a cavity of the lung itself.

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[1] Laennec.: Ausc. méd. vol. i. p. 111. Bourdonnement amphorique.

ii. The voice, cough, and rales.

iii. The cardiac sounds heard through a pneumothorax, phthisical cavity, pneumopericardium, stomach distended with air, or adventitious air-containing cavity below the diaphragm.

II. METALLIC TINKLING. — A sound, first alluded to by Thomas Willis (p. 88), and compared by Laennec[1] to that which is produced in a metallic, glass, or porcelain cup when struck gently by a pin. Metallic tinkling is a single sound, which attends breathing, speaking, coughing, or even mere changes in position of the body. With breathing the sound is mostly intermittent, that is, not heard with each movement; with speaking, the sound is produced more constantly, especially if the patient speak slowly, with a period between each syllable; with coughing, the sound is tolerably constant. Metallic tinkle can sometimes be heard by the patient and bystanders without auscultation.

III. BELL SOUND OR METALLIC RING.—This sign was well known to Laennec.[2] It is the metallic ring of percussion (p. 65) heard by auscultation, that is to say, by putting the ear upon the chest whilst an assistant percusses in the neighbourhood by means of two coins used as plessor and pleximeter. In case of a large cavity full of air, a clear ringing sound will be heard, of a quality best appreciated by comparing it with the sound afforded by the same experiment performed upon a healthy chest : it usually suffices to compare the two sides of the same body.

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[1] Auscultation médiate, vol. i. p. 109.

[2] En auscultant à l'aide du stéthoscope, et percutant

IV. SPLASHING SOUND.—This most venerable physical sign was well known to Hippocrates (p. 88). When a large cavity contains both liquid and air, and the patient is shaken while the physician applies his ear to the chest, a splashing sound is heard. The splash is often heard, and sometimes produced, by the patient himself.

V. PHYSICAL CONDITIONS OF AMPHORIC SOUNDS.—This interesting set of signs, afforded

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en même temps, on entend une résonnance semblable à celle d'un tonneau vide, et mêlée par moment de tintement. Auscultation médiate, vol. i. pp. 111 and 696. Trousseau named the sound "bruit d'airain." Gaz. des hôpitaux, Apr. 4, 1857. There is no reason for thinking that Laennec percussed with coins; nor are they necessary.

by large resounding cavities, has been classed under the head of pleural sounds, because it is in the pleura that the necessary physical conditions are most often met with. These conditions are the following :—

i. A Large Cavity.—The conditions of the metallic ring or bell sound have been already explained (p. 65). The metallic tinkle may be heard in a cavity not larger than a pigeon's egg, provided the cavity open into a large air-tube.[1] The amphoric echo and the Hippocratic splash may occur in cavities of moderate size. The walls of the cavity must be smooth.

ii. The presence of Air in the Cavity.—Succussion splash requires the presence of liquid also. For amphoric echo and bell sound, air alone suffices. Laennec thought that metallic tinkling did not occur unless both air and liquid were present in the cavity ; [2] but liquid is not necessary : the intensest amphoric echo of breathing sounds, or of rales ; the falling of a drop of liquid ; the bursting of a bubble ; and crackling friction sounds, pleural or pericardial ; all are possible causes of metallic tinkle.

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[1] Kolisko, quoted by Skoda. Markham's translation, p. 135.

[2] Auscultation médiate, vol. i. p. 110.

From the foregoing principles it will be now clear, why amphoric echo, metallic tinkle, and bell-sound occur in pneumothorax and in large phthisical cavities: and why the splash may be produced in hydropneumothorax, in large phthisical, suppurative, and gangrenous cavities, and in hydropneumopericardium. Sometimes a distended stomach affords the means of demonstrating all amphoric sounds: the colon will occasionally give succussion splash and bell sound.

APPENDIX TO SECTION II.

There are sundry sounds which, although neither pulmonary nor pleural, depend upon breathing movements.

1stly. Peritoneal Friction, produced between the liver and diaphragm, may be heard all over the chest-walls on the right side.[1]

2ndly. Shoulder-Joint Friction; that is to say, a dry rubbing sound, mostly inspiratory, and heard in the supraspinous fossa, albeit produced in the shoulder-joint. The sound is heard louder as the ear is brought nearer the joint, and is heard loudest over the joint itself. Some positions of the arm stop the sound; some movements produce it.[1]

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[1] Sibson: Medical Anatomy, column 44.

3rdly. Shoulder-Blade Friction, palpable and audible, is sometimes, but seldom, due to friction of the scapula against the ribs, necrosed or not.[2]

4thly. Muscular Rumbling is often heard during auscultation, but, being continuous, cannot be confounded with pulmonary or pleural sounds.

SECTION III.

AUSCULTATION OF THE HEART.

Laennec brought auscultation of the lungs well nigh to perfection, but it was far otherwise with auscultation of the heart. He confirmed the truth declared by earlier physicians, that sound attends the heart's action: he noted that the sound is double: he distinguished murmurs, or unnatural sounds: but when he essayed to go further, and to explain the sounds and the murmurs, he became bewildered and strayed so far even as to deny, in the second edition of his work, what he had maintained in the first, that at least some thrills and some murmurs are associated with valvular obstruction.[1] The errors of Laennec were more serious than those of another man would have been: he led his disciples (and all physicians were his disciples) into a wrong path; nor was it until they discovered where he had strayed, that other discoveries became possible.[2] Three doctrines especially have brought our knowledge of auscultation of the heart into its present satisfactory state: i. the doctrine that the second sound is due to closure of the sigmoid valves;[3] ii. the doctrine of regurgitant murmurs:[4] iii. and the doctrine that murmurs pass in certain directions, and are heard loudest over certain parts of the chest, according to the seat of the valvular disease.[1]

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[1] Gowers: British Medical Journal, Nov. 18, 1876.

[2] Duchenne: Electrisation localisée: 3me édit. p. 951. A case of atrophy of serratus magnus.

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[1] Ausc. méd. 2nd edit. vol. ii. p. 428. Compare 1st edit. vol. ii. p. 316. "Malheureusement pour le cœur, Laennec n'a pas su y enfoncer sa griffe comme il l'a fait au poumon:" Duroziez, Maladies du Cœur, 1891, p. 54.

[2] See an admirable survey of the progress of knowledge during the quarter of a century succeeding Laennec, in Diseases of Heart: 1846: vol. i. p. 31, by P. M. Latham, who lived and practised throughout the whole period.

[3] First taught by Carswell in 1831: see Marc d'Espine: Recherches, etc.: Arch. gén. de Méd. Oct. 1831.

[4] See chapters on Mitral and Aortic Regurgitation. That the blood regurgitates through valves dilated or otherwise diseased, was known long before auscultation was discovered.

ARTICLE I.—HEART-SOUNDS IN HEALTH.

The ear, applied to the heart-region of a healthy person, will perceive that, for each impulse of the heart felt, two sounds are heard. One sound accompanies the impulse, one sound follows it: wherefore the former is called the first or systolic sound (p. 38, note); the latter, the second or diastolic. The two sounds differ in character; the second being sharper and shorter than the first. The first sound is heard loudest over the fourth or fifth intercostal space, just within the left nipple line; the second, over the base of the heart, opposite the third costal cartilage.

There seems to be no doubt that the second sound is due to closure of the semilunar valves. But the question which concerns the cause of the first sound remains much where it was fifty years ago. Let others discuss the two great rival doctrines, whether the sound be due to vibration of muscle or to vibration of valve: and the more so, that murmurs, which constitute our main topic, are chiefly due to another condition, namely, vibration of blood.

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[1] See Williams: Diseases of Chest: 4th ed. 1840: p. 268.

Loudness of the sounds :

i. Whatever weakens the heart weakens the sounds. And weakens the first sound especially : indeed, it sometimes becomes quite inaudible in patients suffering from great vital debility.

ii. But the loudness of the sounds, as we hear them, commonly depends less upon the state of the heart itself, than upon the quantity of material interposed between the heart and ear. Wherefore in fat people, in pulmonary emphysema, and in pericardial effusion, it is a mistake to assume, because the sounds are weak, that the heart's action also is weak. In these cases, the arterial pulse is the true guide to appreciation of the heart's power.

iii. For a similar reason, mere posture, which causes the heart to fall away from the chest-wall in front, weakens the sounds: and therefore they are louder in the upright than in the lying posture.

iv. Unusual loudness of the second sound is common but is insignificant, unless it be the pulmonary second sound which is too loud or accentuated as we say. Accentuation of the pulmonary second sound (heard over the second left interspace) implies an absolute increase of loudness, and not a mere relatively greater loudness as compared with the aortic second sound (heard over the second right interspace). For it may be that the aortic sound is deficient in loudness, the pulmonary sound remaining natural. But this distinction between absolute and relative, no doubt highly rational, cannot always be made in practice. Accentuation of the pulmonary second sound often indicates increased tension of the walls and valves of the pulmonary artery; the greater tension being due to congestion of the lungs, such as occurs especially in disease of the mitral orifice. Moreover, in such cases, this accentuation is probably a sign that the tricuspid valve remains competent, and that the right ventricle contracts with proper force; when valve or ventricle fails, the accentuation may be expected to disappear. Finally, the pulmonary second sound is sometimes strongly accentuated when there are no reasons for suspecting disease of heart or lungs.

ARTICLE II.—HEART-SOUNDS IN DISEASE.

Sounds, heard over the heart-region, and morbid in character, are of two kinds: murmurs and pericardial sounds. The discovery of murmurs was made by Laennec.[1] Several physicians seem to have made the discovery of pericardial sounds; the earliest published reference to them is found in a book written by Collin.[2]

¶ I.—MURMURS.

Any fundamental change in the character of a heart-sound, or any superadded sound heard over the heart region, constitutes a murmur: pericardial sounds excepted. So that it comes to this: we fix a notion in our mind of the healthy heart-sounds: and when we hear a sound over the heart which tallies not with the notion, we say, this sound is either a murmur or a pericardial sound. The diagnosis between these two kinds of morbid sound will be considered under the head of pericardial sounds.

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[1] Auscultation médiate, 1st edit. vol. ii. p. 214.

[2] Des diverses méthodes d'exploration de la poitrine et de leur application au diagnostic de ses maladies, 1824. Pericardial sounds (called craquement de cuir neuf) are spoken of on pp. 64 and 115. Collin was Laennec's chef de clinique; but Laennec disbelieved in pericardial friction sound: Ausc. méd. 2nd edit. 1826, vol. ii. p. 446. P. M. Latham discovered a sound which he deemed to be due to pericarditis, at St. Bartholomew's Hospital in 1826: see Hope: Treatise, 3rd edit. p. 72, 1839. See also Latham's own book, vol. i. p. 101, for a history of the successive discoveries by which endocardial and exocardial murmurs came at length to be distinguished. Latham himself (i. p. 124) assigns the credit of finally establishing the doctrine of pericardial friction to Watson and Stokes.

The physical conditions of murmur have been already explained (p. 94). In health, the respective sizes of the orifices and cavities of the heart are adjusted so that no sound is produced by blood-currents. But whatever contracts an orifice, whatever dilates a cavity, whatever establishes an orifice or a cavity where naturally none should be, will disturb the even flow of blood, and produce vibration and a murmur.

The prime division of murmurs, audible over the heart, is drawn between those which are referable to the heart itself, its walls, orifices, cavities, and contained blood ; and those which are referable to the great vessels next the heart. Hence the distinction between cardiac and vascular murmurs.

Class I.—Cardiac Murmurs.

The physical conditions of murmur are fulfilled, in the case of the heart, at its orifices, and there only : at least we know little or nothing of cardiac murmurs, which are due to any other condition than that of fluid veins produced at orifices. But we cannot affirm this doctrine to be universally true, without much hesitation, when we bear in mind how great the obscurity which hangs over the nature of many heart-murmurs. The orifices of the heart are of two kinds, some are provided with valves, and some are not.

i. VALVULAR ORIFICES.—Each valvular orifice both admits the blood and shuts it off; and whether open or closed, may give rise to murmur. Murmur arises at an open orifice when its size is too small with respect to the cavity beyond: that is to say, the orifice is narrowed, or the cavity is dilated, or both these morbid states concur. Murmur arises at a closed orifice (or say rather, at an orifice which naturally should be closed), when it is not closed, but leaks, and allows the blood partly to pass back into the cavity whence it came. Murmurs produced at an open orifice, and in the natural current of the blood, are called onward, obstructive, or constrictive murmurs: murmurs produced at a closed orifice, and against the natural current of the blood, are called backward or regurgitant murmurs.

ii. NON-VALVULAR ORIFICES—To wit, the

orifices of the venæ cavæ, of the pulmonary veins and the perforations of the auricular or ventricular septum found in congenital malformations, or established after birth. But these non-valvular orifices are very seldom the seat of murmur. Murmur may perhaps arise at the mouth of a large vein when the related auricle is much enlarged, and the rush of blood is strong.[1] An open foramen ovale has sometimes seemed to be a cause of murmur, different, in different cases, as to the place and time where it is best heard. Perforation of the base of the septum ventriculorum may be attended by loud murmur.

Murmurs were once characterised according to their acoustic qualities, whether blowing, filing, rasping, sawing; but these are vain distinctions; in order to render murmurs serviceable in the diagnosis of disease we now regard two only of their properties, namely, their Place and Time.

I.—PLACE OF CARDIAC MURMURS.

In general, a murmur is heard best at that point of the surface of the body which is nearest to the orifice whereat the murmur is generated. So that it becomes important to determine the relation which the orifices of the heart bear to the chest-wall. The Pulmonary orifice lies behind the second left interspace, close to the sternum : the Aortic orifice lies on the same level, behind the sternum : the Tricuspid orifice reaches from the sternal end of the third left intercostal space to that of the fifth right rib ; the Mitral orifice lies on a level with the upper border of the third left cartilage, close to the edge of the sternum, and slightly behind it. And let it be noted that the mitral orifice lies much more deeply than the rest. But murmurs are not always conducted the shortest way to the surface. The conducting power of the tissues interposed between a valve and the chest-wall, and the direction of the blood current, have much to do with determining the point at which a murmur is heard loudest.

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[1] See Markham's case, before cited (p. 43).

i. The influence of superjacent tissues is well exemplified in the case of aortic and mitral murmurs. Both orifices of the left heart lie deep in the chest. The aorta becomes most superficial just above its valves, and is there almost in contact with a good conductor of sound, the sternum : wherefore aortic murmurs are well conducted up and down the sternum, and along the attached cartilages: so that, in fact, an aortic murmur is sometimes heard louder at the xiphoid cartilage than at the second intercostal space. The mitral orifice, buried beneath heart and lung tissues, may be said, for purposes of auscultation, to become most superficial where the cavity of the left ventricle becomes most superficial, that is to say, at, or just above the apex of the heart ; and here, as a rule, mitral murmurs are heard loudest.

ii. The influence of the blood-current (or of convection, as it has been called) is exemplified by both mitral and aortic murmurs. Obstructive and regurgitant aortic murmurs are well conveyed by the blood along the arteries, and a regurgitating murmur may be conveyed by the reflux of blood towards the apex of the left ventricle. Mitral regurgitant murmur is sometimes conveyed by reflux into the auricle, so as to be heard over the third left cartilage.

When the heart is displaced, that is to say when the relation between its valves and certain points of the chest-wall is changed, the place of murmurs is likewise changed. This and many other difficulties and doubts, which we meet with in the exact location of murmurs produced at the different valvular orifices, will be discussed in the second part.

II.—TIME OF CARDIAC MURMURS.

A complete cycle of the heart's action may be divided into four periods. i. Auricular period: not attended in health by any sound: auricles contracting. ii. Ventricular period: accompanied by first sound: ventricles contracting. iii. Arterial period: attended by second sound: aorta and pulmonary artery contracting. iv. The period during which the heart is at rest: unless they are right who maintain Galen's doctrine of an active ventricular diastole. The subject requires a further development, thus:—

	AURICULAR PERIOD.	VENTRICULAR PERIOD.	ARTERIAL PERIOD.	REST.
Relative duration. Pulse=60.	$\frac{1}{10}$	$\frac{4}{10}$	$\frac{5}{10}$	
Valves cuspid	open	shut.		
sigmoid		open	shut.	
Sounds		first.	second.	
Murmurs	præsystolic	systolic	diastolic.	

The time of murmurs is determined in two ways: by reference to the sounds and to the impulse of the heart.

i. By reference to the sounds, murmurs are most accurately timed. When both heart-sounds are heard, it will not be difficult to say that a murmur precedes, accompanies or follows a certain sound. When one sound only is heard, we have to find out whether it be the first or second sound, and this is not always easy. When one sound only is heard at the apex, two sounds are sometimes heard at the base: the sound wanting at the apex is commonly the second. When neither sound is heard at the apex, one sound (especially the second) or both sounds may be heard at the base.

ii. By reference to the impulse. Murmurs which accompany the impulse may be deemed systolic, in the absence of evidence to the contrary. Yet be it borne in mind that the impulse is not strictly systolic, but is also præsystolic and post-systolic (p. 39).

iii. Less trustworthy means of determining the time of a murmur are these. By reference to the arterial pulse, especially in the neck. By reference to a valvular thrill, if any there be, in which case, the thrill and murmur being due to the same vibration, whatever the relation of the thrill to the impulse, such will be the relation of the murmur also.

The time of some murmurs is not constant. This is true of mitral murmurs in particular: a murmur, præsystolic when the patient is first seen, may become systolic before long. But more of this hereafter (pt. ii., chap. 32).

III.—MEANING OF CARDIAC MURMURS.

i. Systolic murmurs referable to Auriculo-Ventricular (or cuspid) orifices indicate reflux of blood: to Arterial (or sigmoid) orifices indicate constriction or obstruction at or just above the orifice, unevenness of the conus arteriosus, dilatation of the aorta or of the pulmonary artery, or perforation of the septum ventriculorum.

ii. Diastolic (and præsystolic) murmurs produced at Cuspid orifices indicate constriction or obstruction of the orifice: at Sigmoid orifices indicate reflux of blood, or dilatation of the vessel above the valve.

But the matter is not so simple as this. Just as the nature of the first sound is more doubtful than that of the second, so is the nature of systolic murmurs more uncertain than that of diastolic (and præsystolic) murmurs. Diastolic (and præsystolic) murmurs indicate in most cases, if not in all, permanent structural (organic) disease. But systolic murmurs are often temporary and unattended by any other evidence of heart-disease: indeed, as will be shown hereafter, their meaning is often doubtful.

IV.—LOUDNESS OF CARDIAC MURMURS.

i. Some murmurs (especially mitral and tricuspid murmurs), which are audible whilst a patient is lying, become much less loud or even disappear when he sits or stands: and some murmurs are louder in the erect posture; but this is less often the case. Wherefore we must examine our patients in both upright and lying postures before we pronounce concerning the presence or absence of murmur.[1] Many mistakes are due to neglect of this rule.

ii. Pressure upon the heart region sometimes makes a murmur weaker. On the other hand, pressure upon the upper part of the sternum or over the pulmonary artery will beget a systolic murmur in some young persons.

iii. The loudness of some murmurs is under the influence of breathing ; thus, a systolic apex murmur may be louder during inspiration than during expiration ; and a tricuspid systolic murmur may become inaudible at the beginning of expiration.

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[1] Elliotson: Lond. Med. Gaz. for 1833: vol. xii. p. 373. Gordon: Posture and heart murmurs: Brit. Med. Journal: March 15, 1902.

iv. Murmurs are loudest sometimes when the heart beats forcibly, sometimes when it beats quietly ; they are sometimes inaudible except at these respective times. In a doubtful case, it should always be a rule (if possible) to end by examining the heart when it has been made to throb by exertion.

v. The loudness of a murmur depends much upon the swiftness of the blood-current ; hence, when the current is weak and slow there may be no murmur, even although the valvular disease is great. Whatever weakens the contraction of the heart weakens the current : hence a murmur often becomes inaudible in the course of infectious fevers, in asystoly, in the dying state, and in other such conditions.

In short, murmurs are apt to be very variable : they come and go, they shift their place and time, in a manner which makes it seldom safe to depend upon a single examination.

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[1] Murmurs (especially musical murmurs) and even oud heart-sounds can sometimes be heard at a distance

V.—Reduplication of Heart-Sounds.

A heart-sound is said to be reduplicated when, instead of being a single sound, it seems to be broken in two, doubled or repeated. Sometimes the sound is completely doubled, that is to say, in the place of one sound there are heard two sounds, with a distinct interval between them. But more often the reduplication is incomplete, or subintrant, that is to say, before the first portion of the doubled sound is concluded, the second begins.

I.—Simple Reduplications.—Most reduplications seem indeed to be nothing more than repetitions of a natural heart-sound, first or second, as the case may be. The component elements of the sound are sundered more or less, and do not exactly concur in point of time. Reduplication of the second sound, for instance, is believed to show that the pulmonary and aortic sigmoids do not close at the same time: if we would fain go further and understand why they do not shut simultaneously, we find ourselves beset by doubts and guesses. The conditions of the first sound being uncertain, it is not safe to attempt an explanation of its

from the chest: see Moore's paper (referred to on p. 89, note).

reduplications. Reduplications are constant or inconstant; they attend every beat of the heart, or some beats only. Constant reduplication sometimes indicates serious disease, for instance, the second sound is often doubled at the base of the heart in the case of obstruction at the mitral orifice. But reduplication is usually inconstant, intermittent, the sound being doubled with some beats of the heart, and not with others. This intermittence is found to bear a close relation with the movements of breathing: so that the first sound doubles at the end of expiration and the beginning of inspiration; the second, at the end of inspiration and the beginning of expiration. The second sound is much more frequently reduplicated than the first.

II. REDUPLICATION MURMURS. — Were reduplications all of this simple kind, they would hardly need much notice. But some seeming reduplications of a heart-sound are indeed murmurs; that is to say, a murmur is added to the heart-sound, or a murmur is split into two sounds, so as to simulate simple reduplications.

False Reduplication of Second Sound.—Bouillaud[1] was the first to describe a bruit de rappel

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[1] Traité: 2me édit., 1841: vol. i. p. 213, and vol. ii. p. 345.

(cantering), that is, a seeming reduplication of the second sound, heard at the apex-beat. A præsystolic murmur also is usually present; whether this be so or not in a given case, the double sound is to be looked upon as being a divided diastolic murmur, and a token of mitral constriction. In fact, cantering sometimes alternates with a diastolic apex-murmur; that is to say, a prolonged diastolic murmur, which is the more constant sign of a mitral constriction, will disappear from time to time, and be replaced by well-marked cantering.

CLASS II.—VASCULAR MURMURS.

There are murmurs which, though heard over the heart region, are believed to be produced not in the heart itself but in a large vessel near it. They are either of uncertain nature or of uncommon occurrence.

i. A murmur, systolic, loudest in the aortic or pulmonary region, often occurs in the state of anæmia. Forasmuch as it is still a matter of debate whether the murmur be valvular or vascular, and whether it be always due to one and the same condition, the further consideration of this topic will be most conveniently deferred to the second part (chapter 40).

ii. A murmur, diastolic, loudest in the aortic region, is sometimes (but seldom) associated with rigidity and dilatation of the aorta, the valves being healthy (part ii. chapter 34).

iii. A murmur, diastolic, loudest in the pulmonary region, is sometimes (but seldom) due to openness of the ductus arteriosus (part ii. chapter 41).

iv. A murmur, diastolic, loudest over the fourth left cartilage, may be due to an opening formed between aortic aneurysm and the pulmonary artery.[1]

¶ II.—PERICARDIAL SOUNDS.

No sound is produced by the movement of healthy pericardial surfaces upon each other.

In diseased states of the pericardium two kinds of sounds may be heard: friction-sounds, and sounds due to the presence of air and liquid in the cavity.

I. PERICARDIAL FRICTION.—It is convenient to consider pericardial friction-sounds with reference to their diagnosis from endocardial sounds. The most important diagnostic characters of friction-sounds are these:—

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[1] Wade: Medico-chir. Trans., vol. xliv. p. 211.

i. The special quality sometimes suffices for the diagnosis, being distinctly rubbing or scraping: but often enough this quality is ill-marked or absent. ii. Friction-sounds are mostly of limited extent, heard over a small portion only of the heart region, especially at its base; they do not follow the laws laid down with regard to the points of greatest intensity, and the conduction of endocardial murmurs. Indeed, there is no part of the heart-region where friction-sounds may not be heard loudest. A loud friction-sound will be heard, not only all over the heart-region but even all over the front of the chest, nay even at the angle of the scapula: though, in these latter cases, there is mostly marked and sudden weakening of the sound so soon as we pass away from its place of origin. Friction-sound, like murmur, may, when very loud, be audible at a distance from the patient's body. Friction-sounds, which at first, on account of their place and their quality, simulate murmurs, sometimes shift their place, so as to become loudest at spots where murmurs are seldom heard loudest. A sound which shifts its place from day to day is pericardial.

iii. Friction-sound is mostly systolic and diastolic, being loudest in the systole; sometimes systolic only, or even diastolic only. A sound, which is at one time systolic and anon diastolic, is pericardial. iv. Friction-sounds are not intermittent like reduplications. v. The unchanged heart-sounds are sometimes heard through friction. vi. Friction-sounds often give a notion of superficiality, be it explained as it may. vii. Palpable vibration sometimes attends them. viii. Their loudness is sometimes increased, sometimes decreased, sometimes stopped, by deep inspiration. ix. The loudness is sometimes increased, sometimes decreased, by pressure. x. Friction-sounds may be modified by change in position of the body. Their place of greatest loudness may be thus made to change: this is very characteristic. Friction-sound sometimes disappears when the patient sits up, possibly on account of a small quantity of fluid which comes forward so as to separate the pericardial surfaces: but the same change in position sometimes removes endocardial murmurs (p. 147). Friction-sound, not to be heard otherwise, is sometimes produced when the patient lies upon his left side, whereby the position of the heart is much changed. xi. In pneumopericardium friction-sound may acquire the character of metallic tinkling.

Whatever roughens the pericardium can produce friction-sound : excessive vascularity, exudations, hæmorrhages, adhesions, and white patches.

II. WATER-WHEEL AND SIMILAR SOUNDS.—In the uncommon condition of effusion of liquid and air nigh to the heart, whether within the pericardium or outside it, peculiar sounds are heard. Sometimes they resemble amphoric pleural sounds, namely, amphoric hum (attending heart-sounds or friction), metallic tinkling, and splashing. Sometimes the sound is like a rale, bubbling or gurgling. Sometimes the sound is clacking or chopping, like the noise made by the floats of a water-wheel (bruit de moulin).[1] All these sounds are alike in this, that they depend upon movements of the heart, are independent of breathing movements, and persist when the breath is held. When the pericardium is uninjured, the sound does not last more than three or four days.

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[1] First described by Bricheteau : Arch. gén. de méd., 4^{e} série, tome iv. : 1844 : p. 334.

APPENDIX TO SECTION III.

There are sounds which, although dependent upon movement of the heart, are neither cardiac murmurs nor pericardiac friction-sounds.

¶ I. Pulsatile Pulmonary Sounds, that is to say, pulmonary sounds produced or altered by movement of the heart or of the great vessels.[1]

i. The respiratory sounds of the healthy left lung may be so affected by movement of the heart as to simulate its murmurs. The conditions of this occurrence are not well understood: pleural adhesions, such as to fix the lung over the pericardium, are often present. The diagnosis lies, not in the quality of the sounds, but in the fact that they concur with a certain respiratory movement, as well as with a certain cardiac movement. They are mostly systolic and inspiratory; less often diastolic, or expiratory. They usually cease to be produced when the breath is held. They are sometimes heard only in the upright posture of the body: but as a rule they are rendered less loud, or they even disappear, on changing the lying posture for the erect.

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[1] Laennec: Auscultation médiate, vol. ii. p. 446, describes altered breathing sounds and crepitation.

ii. The respiratory sounds of a cavity (phthisical, bronchial, or pneumothoracic), may be altered by the movements of the heart or aorta in a similar manner. These false murmurs also are mostly inspiratory and systolic, or systolic and diastolic; seldom expiratory. They are heard best over the cavity, but they are sometimes conducted far, even over the whole chest. And, in the case of phthisical cavity, even along the windpipe to the mouth, so as to be heard at a distance from the patient, when the mouth is opened widely (pulse-breath, see note). The cavity is either close upon the heart and aorta, or is connected with them by a solid mass (such as solidified lung, or enlarged lymphatic glands), which is apt to convey the cardiac movements. In cases of phthisis, a strong aortic impulse can sometimes be felt where the false murmur is heard: for instance, in the third right intercostal space, close to the sternum.[1]

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[1] By listening to the open mouth of a person breathing quietly, a gentle puffing sound can sometimes be heard to attend each beat of the heart during expiration. This Pulse-breath was described first by Radclyffe Hall (Med. Chir. Trans. for 1862: vol. 45, p. 167), and afterwards by David Drummond (Brit. Med. Journ., Oct. 21, 1882: p. 773). Drummond auscultates with a peculiar stethoscope, one end being put into the patient's mouth:

iii. Rales also may be produced, and especially crepitation. It is systolic, and is most likely due to the entry of air into vesicles partly or wholly collapsed, the movement of the heart causing a local inspiration in the portion of lung which lies upon the pericardium. The crepitation ceases to be produced when the breath is held after a deep expiration.

¶ II. Pulsatile Friction Sound generated in the pleura, of both respiratory and cardiac rhythm, sometimes is heard. Friction heard behind or alongside the sternum, from the second to the sixth rib, is most likely pericardial. When a friction sound is heard at other parts of the heart-region, the diagnosis is not easy: pleural friction usually ceases when the breath is held, but this is not always the case; and there is no doubt that true pericardial friction may sometimes be stopped in that manner.

¶ III. By the name of Metallic Jingle,

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by this means a pulse-breath (or oral whiff) can be heard in healthy people when the heart is beating forcibly. The diseases which favour the occurrence of pulse-breath are aneurysm of the aorta, and cavity within the lung. The sound, when loud, can be heard by auscultation of the trachea.

Laennec[1] meant a sound which is heard when the stethoscope is applied to the chest, whilst some bony part near by (such as the clavicle or spine) is percussed. When the palm of the hand is put over the ear and the back of the same hand is tapped, we hear a loud metallic jingle. A sound of the same kind is sometimes heard when we listen to a heart which is beating forcibly; in this case, it is commonly supposed that the stroke upon the ribs causes the sound. For my own part, I suspect that resonance of the meatus auditorius externus has much to do with the production of a metallic jingle.

Auscultation as applied to the large vessels of the mediastina will be treated of hereafter.

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[1] Auscultation médiate, vol. i. p. 114, and vol. ii. p. 445; cliquetis métallique.

> "Magis auscultandum quam audiendum censeo."
>
> PACUVIUS

CHAPTER VI.

APPENDIX TO PART I.

SECTION I.

AUSCULTATION OF ARTERIES.

TWO kinds of sound are heard by auscultation of arteries: conducted sounds and murmurs produced in the part ausculted.

I. CONDUCTED SOUNDS.—The heart's sounds and murmurs, especially the second sound, and aortic or mitral murmurs, are conducted along the arteries; not usually, however, farther than the carotids and subclavians. Yet very shrill diastolic murmurs produced at the aortic orifice, may sometimes be heard so far away as in the radials.

II. MURMURS PRODUCED IN THE SPOT AUSCULTED.— These are either spontaneous or factitious.

¶ I. Spontaneous Arterial Murmurs.—These are aneurysmal or subclavian.

i. Aneurysmal.—The systolic murmur which is sometimes produced in an aneurysm, is believed to be due to formation of a fluid vein at the mouth of the aneurysm. Formation of a fluid vein requires that the cavity containing fluid blood be considerably wider than the mouth of the sac, and that the blood current be swift enough.

ii. Subclavian.—A systolic murmur, seated in the subclavian artery, is sometimes heard below the clavicles, mostly on the left side. The murmur is often inconstant or temporary. Forasmuch as it cannot be said to denote any form of disease, it seems unnecessary to discuss the doubtful conditions under which the sound is supposed to occur.

¶ II. Factitious Arterial Murmurs, due to compression of an artery by the stethoscope, are systolic or diastolic.

i. Systolic Murmur. — In healthy persons, a slight compression of the larger arteries will generate a soft murmur, systolic with reference to the left ventricle. In some forms of disease, notably in hypertrophy of the left ventricle, and in chlorosis, firmer pressure makes the murmur harsh and whizzing. Moreover, under these conditions, a murmur is producible in smaller arteries ; such as the volar.

ii. Diastolic Murmur has long been known ;[1] but Duroziez was the first to study it more particularly.[2] It is best heard in the femoral artery, and attends a certain degree only of pressure : a degree which must be discovered in each case by varying the amount of force used to compress the vessel. A loud systolic murmur precedes the softer diastolic sound. It is often present in cases of aortic regurgitation ; and is probably due to reflux of blood along the artery, during the ventricular diastole. A certain amount of pressure upon the artery above the spot auscultated renders the systolic murmur louder ; the reason being that a second fluid vein is thus produced. The diastolic murmur is rendered louder by compression of the artery below the spot auscultated, probably because diastolic reflux is favoured thereby. Yet double femoral murmur sometimes occurs apart from any signs of valvular disease : in this case the condition has been supposed to lie in a highly dicrotic pulse ; the diastolic murmur corresponding with the secondary pulse-wave.

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[1] Bouillaud : Traité : 2me éd., 1841, vol. i. p. 228.

[2] Duroziez : Arch. gén. de méd. : series v. vol. xvii., 1861. To contemplate the vast extent of literature devoted to this very insignificant topic is amazing and amusing.

It is necessary to bear in mind that diastolic murmur, produced at the aortic orifice, is sometimes conveyed to great distances along the arteries. But diastolic murmur of this kind requires no compression of the artery to make it audible.

SECTION II.

INSPECTION OF VEINS OF NECK.

What examination of the arterial pulse is to detection of diseases of the left heart, such is examination of the veins to detection of diseases of the right heart.

Inspection of the veins of the neck is directed to two points; the fulness of the veins, and the movements which the contained blood undergoes.

¶ I.—FULNESS OF THE VEINS.

In health the external jugular alone is visible, and even that vein, oftentimes, only in the lying posture. In disease both veins are sometimes dilated to the size of a finger. Overfilling of the veins is either transitory or permanent.

i. Transitory overfilling accompanies powerful expiratory movements; which produce such an amount of pressure within the intrathoracic veins, that the valves at the mouths of the jugular veins are shut, and the blood flowing down from above cannot pass into the innominata. Inspiration reverses all this; the veins are emptied, and collapse. Repeated transitory overfilling of the veins is followed at last by permanent dilatation of them, a fact seen in patients who suffer from chronic pulmonary catarrh. If in these persons when there is no cough and the veins are invisible, we place our finger just above the clavicle so as to obstruct the external jugular vein, it at once swells up and manifests the amount of its dilatation; which may be taken as a mark of the degree to which the patient's tissues have suffered in consequence of cough.

ii. Permanent overfilling of the jugulars is mostly associated with overfilling of the right auricle; but, obviously, any obstruction to the upper vena cava, or to the innominata (by compression, thrombosis, or stricture) will have the same effect.

¶ II.—MOVEMENTS WITHIN THE VEINS.

Besides the respiratory movements which have just been described, the blood within the veins often undergoes movements which are dependent upon the heart's contractions. The most obvious kind of venous pulsation is that seen in veins visibly distended. There is a degree of distension which checks pulsation : for this reason, pulsation sometimes attends only the upright posture of the body, when the veins are less distended ; and disappears in the lying posture, when the distension becomes greater. Moreover, pulsation of the jugular veins can often be seen, when the veins themselves are quite invisible ; just as the pulse at the wrist can be felt, but not the artery of a healthy man. There is usually no difficulty in distinguishing this venous pulse ; it is less decisive and definite, more fluttering and quivering (dicrotous, tricrotous) than the arterial pulse ; and the finger, put upon the pulsating vessel, stops, but seldom feels, the pulsation. Venous pulsations are præsystolic or systolic.

i. Præsystolic pulsation[1] of the jugular veins may be sometimes seen in persons who are free from disease of the heart. Anæmia and the horizontal posture favour this pulsation. It is followed by a systolic emptying of the vein.

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[1] First noted by Popham : Dublin Quart. Journ. Med. Sc. 1855 : vol. xix. p. 469.

This præsystolic pulse is believed to be due to sudden arrest of the onward venous flow during the auricular systole: the systolic emptying of the vein to a sudden auricular diastole. Præsystolic jugular pulsation in disease may be due to regurgitation of blood into the vein.

ii. Systolic pulsation in the veins is due to the ventricular systole, indirectly or directly. A direct systolic pulsation signifies a reflux of blood out of the ventricle; the tricuspid valve being incompetent. When the valve is competent, we may call the pulsation indirect.

a. Indirect systolic pulsation is explained in different ways, and perhaps is not always due to the same cause. It has been supposed that the tricuspid valve is raised, by the ventricular systole, into a sort of dome, convex towards the auricle; and hence an impulse backwards to the blood in the venous system; counteracted, however, unless the overfilling of the veins be great, by the diastole of the auricle. It has been suggested that in some cases the systolic filling of the aorta compresses the distended intrathoracic veins, and thus produces a movement in the jugulars.

β. Direct systolic pulsation, being due to propulsion of a wave of blood from the right ventricle into the jugular veins, requires that both tricuspid and venous valves be incompetent.[1] It is easy to ascertain whether the venous valves are competent or not; namely, by compressing the veins in the upper part of the neck, and observing whether they are filled with blood from below or not. But it is not so easy to determine reflux through the tricuspid valve; that is, to distinguish between direct and indirect systolic pulsation. The difference is only one of degree: pulsation is less marked when indirect than when the tricuspid valve is incompetent: for in the latter case blood is pumped out of the ventricle into the veins under strong pressure. In both cases, however, systolic jugular pulsation is an important sign of an affection of the right heart: for even if, in a given patient, there are no grounds for supposing the tricuspid valve to be incompetent, the pulsation indicates an engorged state of the right chambers, such as will dilate them, if it continue. For this reason, in mitral disease, jugular pulsation often precedes any evidence which percussion affords of secondary distension of the right heart. When the pulse is very strong, it is sometimes palpable, or even thrilling.[1]

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[1] Joh. M. Lancisii, De motu cordis et aneurysmatibus. Lugd. Bat. 1740. Propos. lvii. "Inquirere mechanicam rationem, ob quam in dilatationibus radicis Cavæ, Auriculæ, et Ventriculi dextri, ipsæ venæ Jugulares vicissim dilatentur, fluctuent, mirisque modis agitentur, et concidant." The cause is asserted to be a regurgitation of blood through the tricuspid valve. Lancisi refers to Hombert as having made the same observation in a paper published in the Proceedings of the Parisian Academy of Sciences in 1704. See also Stokes: Diseases of Heart, 1854: p. 199.

By the sphygmograph applied to jugular veins, pulsating in cases of diseased heart, the venous pulse is found to be dicrotous; but dicrotous in a manner different from the arterial pulse. The venous dicrotism occurs in the rise of the blood-wave. The first or smaller impulse coincides with the auricular contraction; the second or chief impulse with the systole of the ventricles. Sometimes there is an impulse at the very end of the fall of the pulse-wave: supposed to indicate repletion of the cavities of the right heart, a sudden stop being put to the entry of more blood.

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[1] In the case of rupture of aortic aneurysm into the vena cava superior, systolic pulse and thrill are present in the veins of the neck

Fig. 13.

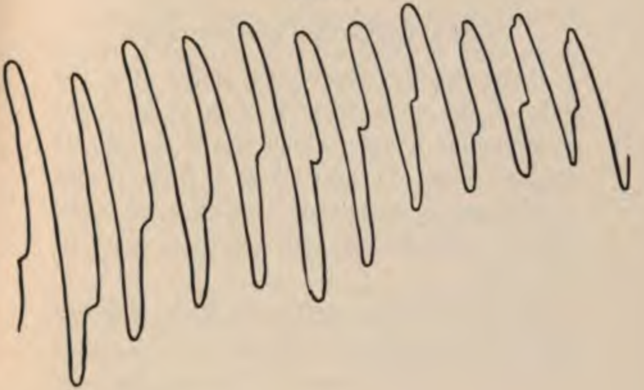
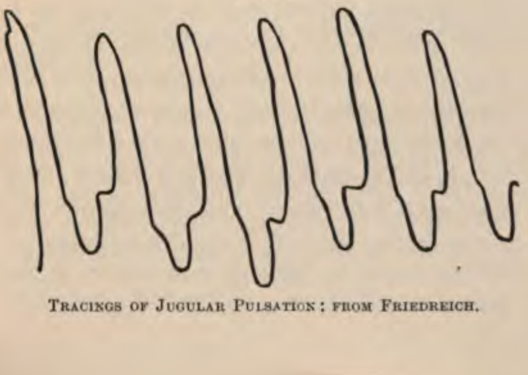


Fig. 14.



TRACINGS OF JUGULAR PULSATION: FROM FRIEDREICH.

SECTION III.

AUSCULTATION OF VEINS OF NECK.

By means of a stethoscope placed upon the side of the neck, there is often to be heard a humming sound; which was first referred to the veins by Ogier Ward.[1] This venous hum is usually continuous; but other murmurs, which are intermittent, are sometimes heard in the veins.

¶ 1. The Continuous Venous Hum, although especially well heard in chlorotic patients, occurs in many healthy persons. Pressure with the stethoscope is doubtless a frequent cause of the murmur. But a venous hum, in chlorotic persons, is independent of pressure, and is believed to depend upon anatomical conditions. The internal jugular vein is adherent, at its lower end, to the cervical fascia, in such a manner that, when the venous system shrinks in capacity and adapts itself to a lessened bulk of blood the part of the vein spoken of cannot shrink, and so becomes relatively dilated: hence a sonorous fluid vein.[2] The more rapid the flow of blood the louder the hum : hence it is louder in the erect than in the lying posture ; and is stopped by whatever produces stagnation of blood in the vein. Hence, also, in most cases, although the murmur is continuous, yet it is subject to rhythmical increase of loudness ; both during the inspiratory draught of blood from the veins, and during the ventricular systole, that is, during the auricular diastole, when the venous current begins again to flow freely.

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[1] On the bruit du diable. Lond. Med. Gaz. 1837, p. 7.

[2] Chauveau: Gaz. méd. de Paris: 1858: pp. 340 et seq.

¶ II. Intermitting Venous Murmurs, præsystolic, systolic, diastolic, have been described, but many are doubtful and none of the least importance.[1]

SECTION IV.

EPIGASTRIC PULSATION.

¶ I. Pulsation of the epigastrium depends upon—

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[1] See Parrot : Arch. gén. de Méd., June, 1867, p. 649. Also Friedreich, Krankheiten des Herzens, 2nd edit. p. 96 ; Ringer and Sainsbury : Lancet for 1891, vol. ii. pp. 1,212 and 1,268, and for 1892, vol. i. pp. 740 and 790. Murmurs heard in the femoral veins have been studied by Schreiber (Deutsch. Arch. für. klin. Med. vol. 28, p. 243), Beau (Traité, p. 416), and Friedreich (Berlin klin. Woch. for 1874, No. 48, p. 611).

I. Conduction thereto of the impulse of the heart: i., when it is beating more strongly than usual: ii., when it is dilated: iii., when it lies lower than natural; a condition mostly due to depression of the diaphragm (p. 175); iv., when it is displaced to the right (p. 42).

II. Pulsation of the abdominal aorta, or of the cœliac axis, or of an aneurysm, or of a tumour seated upon the abdominal aorta.

III. Regurgitation of blood into the hepatic veins, consequent upon dilatation of the right heart, and causing systolic pulsation of the liver; a phænomenon first observed by Friedreich.[1] Hepatic pulsation was noted so long ago as the time of Senac; but the older physicians believed that the liver merely conveyed an impulse from the heart, vena cava, or aorta. The distinction between the two kinds of pulsation, conducted and refluent, depends upon the following considerations. i. Whether the heart or any adjacent organ be pulsating powerfully and extensively: it is seldom difficult to distinguish between the slow, gentle venous pulse, and the quick, strong heart impulse. ii. Whether there be signs of tricuspid disease, and, in particular, whether the jugular veins afford a regurgitant pulse. iii. When paracentesis for ascites has been performed and the abdominal walls are left very flabby, the liver can sometimes be grasped, and felt to expand, like an erectile tumour, at the time of impulse.

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[1] See Mahot: Des battements du foie: Paris, 1869.

¶ II. Recession of the epigastrium, systolic, occasionally simulates pulsation, and when well marked is probably due to pericardial adhesions.[1]

SECTION V.

POSITION OF DIAPHRAGM.

In many diseases of the chest, it becomes an important element in diagnosis to ascertain the position of the diaphragm. Strictly speaking, we determine the lower limits of the lungs and heart, the upper limits of the liver, spleen and stomach, and deduce the position of the diaphragm from these data. For this purpose all the means of physical examination are more or less serviceable, but percussion is especially useful.

¶ I. In Health.—By inspection, the position which the diaphragm held before puberty may be roughly determined (p. 16). By palpation, the position of the heart's apex-beat, and the point where vocal vibration ceases, are ascertained. By percussion, the lung, at the end of an ordinary inspiration, is found to reach, in the sternal line the lower border of the sixth rib, in the nipple line the upper border of the seventh rib, in the axillary line the lower border of the seventh rib, in the scapular line the ninth rib, and in the spinal groove the eleventh rib. The lung, during quiet breathing, fills not the whole pleural cavity, but leaves it unoccupied at the part most distant from the bifurcation of the bronchi, namely, at the semicircular channel formed by the chest-wall and the diaphragm where it shelves downwards to be attached to the ribs. This unoccupied portion of the pleural sac has been named the complemental space; there the costal and diaphragmatic pleuræ are in contact.

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[1] Associated, in Copland's case (Dict. pract. Med. vol. ii. p. 214), with cup-shaped depression of the sternum.

¶ II. IN DISEASE.—The disease may be such as to afford an obvious impediment to physical diagnosis, by destroying the resonance of the parts above the diaphragm. Which is the case in solidification of the lower lobe of the lung, and in liquid pleural effusion. When the lung is solidified, vocal thrill, if present, will assist us, inasmuch as it fails rapidly beyond the pulmonary region. When liquid is present in the lower part of the pleura, it is impossible to do more than guess where the level of the diaphragm may be. The diaphragm lies high in :—contraction of the lung, distension of the abdomen, abdominal tumours or intumescence, paralysis of the diaphragm. The diaphragm lies low in :—hypertrophous emphysema of the lungs, pleural effusions, dilatation of the heart, pericardial effusions, intrathoracic tumours, spasm of the diaphragm. A greatly enlarged heart or an abundant pericardial effusion may depress the diaphragm so much as to produce a tense swelling in the epigastrium.[1] Depression of the right wing of the diaphragm sometimes depresses the right lobe of the liver in such a manner that the left lobe is tilted upwards and raises the apex-beat of the heart.

SECTION VI.

POSITION OF MEDIASTINUM.

The position of the mediastinum is determined in the same manner as the position of the diaphragm, namely, by ascertaining the position of the organ which is most intimately connected with the mediastinum, that is the heart. By palpation we discover the position of the apex-beat of the heart: by percussion we are enabled to confirm the notions acquired by palpation, and to map out the position assumed by the heart. The mediastinum is displaced in unilateral pulmonary or pleural disease; and the displacement is either towards or away from the seat of disease. The mediastinum is displaced towards the seat of disease when one lung is shrunken: this is especially seen in phthisis, cirrhosis, and collapse, but also, to a less extent, in an adherent pleura. The mediastinum is displaced away from the seat of disease, in unilateral pleural effusions of liquid or gas. When the effusion is liquid we possess an additional means of determining the position of the mediastinum, to wit, percussion of the sternal region above the heart. The upper part of the sternum naturally yields a clear resonance: under the pressure of a copious liquid effusion into either pleura, the mediastinum bulges so much towards the unaffected side, as to afford absolute dulness to percussion in the sternal region, and even somewhat beyond it. Intrathoracic tumours sometimes displace the heart.

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[1] Auenbrugger : Inventum Novum, § 46.

The displacement of the mediastinum, which takes place in unilateral pleural effusions, is not at first due to pressure exerted by the effusion.[1] The lungs, in health, are in a state of distension which is kept up by excess of atmospheric pressure from within; the thoracic walls bearing off the atmospheric pressure from without. The elasticity of the lung is continually striving to overcome this distension, as is manifested by the relaxation of the lung which ensues when the internal and external atmospheric pressures are equalised. The distended lungs of the healthy chest, with their elasticity in full play, drag upon the mediastinum; which, however, maintains its natural position, because the forces on both sides are equal. But if the elastic traction of one lung be destroyed by relaxation or collapse, the other lung, no longer counterbalanced, itself relaxes as much as possible, and draws the mediastinum away from the middle line.

SECTION VII.

OTHER VASCULAR MURMURS.

Very probable though the occurrence of murmurs within the vessels of the lungs may be, yet the fact has not been often proved. In the right supraspinous fossa of a phthisical patient, Gerhardt heard, beside bronchial breathing and ringing rales, a systolic whiff; which was explained, post mortem, by the existence of a dilated branch of the pulmonary artery, running across a cavity, and expanded in one spot to a small aneurysm.[1] Between the scapulæ and vertebræ Immermann once heard systolic murmurs, due to constriction of the pulmonary arteries at their entry into the lungs, and just beyond; constriction caused by induration of the pulmonary tissue.[2] Murmurs of similar character, and presumed to be of similar origin, have been heard by many other persons.

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[1] Powell: Med. Times and Gaz., Jan., Feb. 1869. Also: Med. Chir. Trans., vol. 59, p. 165: 1876.

Murmurs, having the character of vascular murmurs, are often heard below the clavicles and behind the manubrium sterni. These murmurs seem to be sometimes arterial, sometimes venous; but it is seldom possible to attain any certainty respecting the particular vessel in which they arise.

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[1] Lehrbuch, 3rd edit. pp. 218, 270.

[2] Deutsch. Arch. für klin. Med. vol. v. 1869.

SECTION VIII.

PUNCTURE OF CHEST.

Since the discovery of auscultation, no more important addition has been made to our means of physical examination than the method of probing the chest by puncture.[1]

The end proposed is to detect collections of liquid within the chest; pleural effusions, pericardial effusions, and hydatid cysts.

The instrument employed is a small glass syringe, holding half a drachm or a drachm, and fitted with a hollow steel needle an inch and a half or two inches long, and as fine as is consistent with due strength.

The place chosen for puncture is that where liquid is supposed to be: due regard being paid to the anatomy of parts within, so as to beware of wounding the heart, diaphragm, or large vessels.

i. When a pleural effusion is suspected, the puncture should be made where the signs of disease are most marked. But, if possible, let the spot chosen be somewhere between the angle of the scapula and the edge of the pectoralis major, and not much below the nipple level. The bare suspicion of a pleural effusion, however small it seem to be, is a sufficient reason for exploring the chest by puncture, inasmuch as we know that to pierce the lung with a fine needle is harmless. Indeed I may say, as the outcome of a very large experience, that I do not remember a single patient to have been left the worse for a puncture made to detect a suspected pleural effusion.[1]

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[1] The practice of puncturing the chest by a fine needle and for the purpose of diagnosis, as distinguished from paracentesis thoracis, was introduced by Thomas Davies: see his Lectures on diseases of the lungs and heart: London, 1835: p. 344.

ii. Puncture is of much less use in the diagnosis of pericardial effusion. We dare not puncture upon the bare suspicion that a pericardial effusion may be present; we require that its presence should first of all be rendered most highly probable by other means of physical examination. But these other means do not always enable us to distinguish between pericardial effusion and dilated heart; yet, if we resort to the exploring needle to clear up our doubt, we know that puncture of a dilated heart (especially of a dilated auricle) may kill the patient. Wherefore the greatest care is necessary. The rule is to puncture through the fifth left interspace an inch from the edge of the sternum. But the operator must use his discretion; by reference to published cases it will be seen that puncture has been successfully made in the third, fourth, fifth, sixth or seventh left interspaces, and in the third or fifth right interspaces.[1]

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[1] But see Lancet for Dec. 2, 1905, where cases are cited of four patients who died immediately after exploratory puncture, and three of them from pulmonary hæmorrhage. I have heard of a case in which an intercostal artery was punctured and the patient died from bleeding into the pleural cavity. But this is an accident extremely rare.

iii. When a hydatid cyst is suspected, the rule seems to be to pierce the lung with a long needle: at least this is the practice in Australia, where the disease is common.[2]

The needle may enter a phthisical or other suppurating cavity within the lung, and the syringe draw off a little pus or muco-pus. When the diaphragm is much pushed upwards by a large abscess between it and the liver, and when, as is often the case, an empyema of the lower part of the right pleural cavity also is present, the exploring needle sometimes passes right through pleural empyema and diaphragm, so as to obtain pus from the abdominal empyema only. Subcutaneous emphysema sometimes follows puncture of the chest, either in old pleural adhesions or in pneumothorax.

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[1] Samuel West: Med. Chir. Trans. vol. 66, p. 235.

[2] S. Dougan Bird: On hydatids of the lung. 2nd edit. Melbourne, 1877. Says that puncture for hydatids of the lung was first described by R. F. Hudson, in the Australian Med. Journal for April, 1861. Aspiration of a pulmonary hydatid is dangerous, and even exploratory puncture is not quite free from risk of suffocating the patient in consequence of the hydatid fluid entering the bronchia.

SECTION IX.

CONDUCTION OF PULMONARY AND PLEURAL SOUNDS.

Unnatural respiratory sounds, heard by auscultation, are sometimes conducted to a distance from their place of origin. This is true of unnatural breathing sounds, of rales, and even (but seldom) of friction sounds. Thus a sound arising on one side of the chest may be heard on the other side, arising at the apex may be heard lower down, arising behind may be heard in front, arising in the larynx or trachea may be heard all over the chest. The sounds lose in loudness by conduction, and this fact is the guide to their place of origin: where the sound is loudest, there it arises. For the identification of a conducted sound with the original sound it is necessary that they both possess the same qualities, although the former be the less loud.[1]

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[1] See Skerritt: Brit. Med. Journ., Nov. 22, 1884: p. 1005.

AUSCULTATION OF THE ŒSOPHAGUS is of very little use, but has been practised in two ways. i. The patient's chest is auscultated along the course of the œsophagus, whilst he is swallowing a mouthful of liquid (see W. Hamburger: Klinik der Oesophaguskrankheiten: 1871. Also, Mackenzie, Dis. of Throat and Nose: 1884: vol. ii. p. 7). ii. A flexible tube, passed down the gullet, is attached to a stethoscope; whereby neighbouring organs, especially the heart, can be listened to through the œsophagus (see Richardson; Asclepiad, No. 36: 1892: p. 371. Also, Hoffmann: Centralblatt für klin. Med. Dec. 3, 1892).

PART THE SECOND.

CHAPTER I.

PULMONARY CATARRH, OR BRONCHITIS.

§ I. RESPIRATORY sounds weakened, and rales: these are the signs of catarrh.

¶ I. Weakening of respiratory sound is general or local: that is to say, the whole of both lungs, or only a part of them, is so affected. i. Local weak breathing is by far the commoner condition; is due to the presence of mucus in the tubes; may amount to complete suppression of sound; is usually movable in seat; and of short duration at any spot. ii. General weak breathing is due to swelling of the mucous membrane, or to weakened respiratory movements.

¶ II. Rales are of two kinds, sonorosibilant and mucous. Each kind hides the respiratory sound more or less. i. Sonorous and sibilant rale indicates local incomplete obstruction of the larger air-passages by mucus. It often happens that this sound arises in the larynx and is thence conducted through the lungs: in which case, a cough, by removing the mucus, removes the rale. ii. Mucous rale indicates the presence of mucus in the tubes of the lung itself. The seeming size of the rale is usually proportionate to the size of the tube wherein the rale is produced. Mucous rale requires that the respiration be fairly vigorous: when the ebb and flow of air are much impeded the rale is imperfectly developed (obscure rale): a deep breath will sometimes change obscure into distinct rale. When no rales are heard, even although the expectoration is profuse, the excessive secretion probably comes from the largest windpipes.

§ II. i. Uncomplicated pulmonary catarrh is bilateral, affecting both lungs: mucous rale, when present, is most abundant behind and at the bases of the lungs, or indeed exists there only. Yet now and then, and in simple bronchitis too, abundant rale is heard for a short time in one lung only. However, persistent localisation of the signs of catarrh to one lung, or to a portion of lung other than the base, is an almost sufficient proof that the catarrh is determined upon the part affected by some additional disease. ii. Severe pulmonary catarrh is attended, especially in very young or rickety children, by signs of inspiratory dyspnœa (p. 36), namely recession of the epigastrium, of the ribs and cartilages below the nipples, and of the supraclavicular spaces. iii. In the same class of patients, acute emphysema or insufflation of the lungs is sometimes rapidly produced; it is indicated by bulging of the front of the chest.[1] iv. The percussion-note is often impaired, for a few days, even over the whole of a lung; more commonly over a part only, especially the apices, the lower lobes, and the middle lobe of the right lung: associated collapse being the usual cause. On the other hand, in both children and adults, patches of unnaturally clear resonance may be met with; probably due to local relaxation of lung tissue. v. When catarrh is complicated by scattered solidifications of the lungs (especially lobular pneumonia and miliary tuberculosis) the rales acquire a sharp reverberating quality. Dilated tubes produce the same effect, but this lesion is very uncommon compared with scattered consolidations. vi. Deformities, produced by catarrh, are sometimes permanent:

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[1] "Pejorante passione thorax etiam extantior fiet." Caelius Aurelianus; Acut. morb. lib. ii. cap. 27, ¶ 144.

and have been already described under the names of pigeon breast, cupping of the lower part of the chest in front, and bulging of the upper part of the chest in front.

§ III. i. Œdema of the lungs, blood and diphtheritic exudations in the air-tubes, and miliary tuberculosis, cannot be distinguished from simple catarrh by physical signs alone. ii. Pneumothorax is simulated when the main bronchus of a lung is completely plugged by mucus; an accident sometimes met with in the stupor of cerebral diseases, sometimes occurring rather suddenly in the course of simple bronchitis. The breath-sound is suppressed over the greater part or the whole of one side, and the percussion-note is clear. But the displaced mediastinum and amphoric signs of pneumothorax are wanting. iii. Plugging of a main bronchus by anything but mucus is usually a severer condition: the signs being the same, the diagnosis depends on other circumstances.[1] iv. The rale of pleurisy is sometimes mistaken for a simple catarrhal rale: pleuritic rale however is mostly unilateral.[2]

The physical signs of that form of chronic bronchitis which is characterised by expectoration of fibrinous casts, are sometimes the same as those of catarrhal bronchitis, local or universal. But very often there are no morbid signs at all, the disease being of small extent. On the other hand, when the bronchial exudation is considerable, collapse of a large portion of lung ensues, with corresponding physical signs.[1]

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[1] See chapter ix.

[2] See chapter x.

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[1] See chapter viii.

CHAPTER II.

PULMONARY ŒDEMA.

THE simplest kind of pulmonary œdema is that which occurs acutely in the course of renal dropsy: it is to this form of œdema that the following remarks especially apply. The other kinds of œdema of the lungs are usually more or less complicated with other pulmonary lesions, such as the chronic bronchitis and brown induration of cardiac dropsy.

§ I. The physical signs of pulmonary œdema are chiefly those which denote the presence of thin liquid in the air-passages.

¶ I. The respiratory sound is hidden by the rales. Bronchial breathing sometimes is heard in compact œdema, apart from any compression of the lung by hydrothorax.

¶ II. Mucous rales, small in size, often sharp and reverberating. True crepitation, either in patches here and there, or much more extensive so as to involve the whole of one or both lungs, is sometimes present.

§ II. i. Pulmonary œdema is bilateral sooner or later, but it may attack one lung some hours before the other. ii. Hydrothorax is a complication almost constant; and also bilateral, unless obliteration of one pleural cavity by adhesion render the effusion necessarily unilateral. iii. Great inspiratory dyspnœa is sometimes present; attended, it may be, by extreme recession of the infra-mammary regions, even when hydrothorax also is present. Inflation of the front of the lungs often ensues, such as to cause the heart's dulness to disappear. iv. Percussion-note unaffected: or somewhat diminished in resonance: or unnaturally clear in patches where the subjacent lung happens to be relaxed in consequence of the œdema or hydrothorax. Dulness to percussion, at the bases of the chest in proportion to the hydrothorax: yet considerable pleural effusion will sometimes yield no percussion dulness, even when the diaphragm and liver are depressed so much as to distend the abdomen. v. Dilatation and pulsation of the jugular veins may sometimes be seen.

§ III. The diagnosis of pulmonary œdema from pulmonary catarrh depends more upon symptoms than upon physical signs. Compact or solid œdema affords dulness to percussion and bronchial breathing, and thus simulates pneumonia.

CHAPTER III.

PULMONARY CONGESTION.

§ I. THE only physical sign which has been supposed to indicate simple pulmonary congestion consists in accentuation of the cardiac second sound over the second left interspace close to the sternum ; in other words, the pulmonary second sound is louder than the aortic. It is in diseases of the mitral orifice that this sign is most often met with, and it is of some value when taken in this connection (p. 136).

§ II. Passive pulmonary congestion is usually associated with catarrh and its physical signs. Other complications, also common, are collapse, œdema, hydrothorax, and hæmorrhagic infarctus.

§ III. Brown induration of the lungs is a consequence of chronic congestion. Some impairment of percussion resonance, especially over the lower lobes and on the left side, is the only sign relative to this lesion.

CHAPTER IV.

PULMONARY HÆMORRHAGE.

§ I. THE physical signs of a moderate bronchial hæmorrhage are simply rales due to the presence of blood in the tubes : when the blood is expectorated as fast as poured out, there will not even be rales.

§ II. Hæmorrhagic solidification of the lung, when extensive enough (not less than three inches in diameter at the surface) yields the physical signs which would be expected, namely, dulness to percussion and bronchial breathing. Hæmorrhagic infarctus is usually complicated by chronic catarrh and congestion, with their consequences.

CHAPTER V.

PULMONARY EMPHYSEMA.

BY pulmonary emphysema is meant progressive dilatation of the air-sacs and destruction of their septa, associated with increase in the bulk of the lung; hypertrophous emphysema.

§ I. The signs indicate enlargement of both lungs. i. Bilateral enlargement of thorax. ii. Depression of diaphragm, involving depression of heart, liver, spleen, and stomach: cardiac epigastric pulsation is a very early sign. iii. Extension of lung in front of heart: whereby the area of superficial cardiac dulness is diminished or abolished, and the heart's impulse and sounds become enfeebled. iv. Bulging of lungs above clavicles, especially during cough or powerful expiration.

§ II. i. Emphysema is always bilateral, unless one lung be otherwise diseased so as to forbid its expansion. When one lung is indurated, or greatly collapsed from unyielding pleural adhesions, it is common to find emphysema of the other lung. ii. The respiratory movements tend to assume the characters described under the name of non-expansive inspiration and expiratory dyspnœa. iii. The percussion-note tends to fall in pitch, that is to say, to become tympanitic; the muffling mostly remains unchanged, or is even increased, although sometimes the note becomes clear in places. iv. The respiratory sound is usually weakened, in consequence of non-expansive inspiration. And the expiratory sound is often greatly prolonged, in consequence of expiratory dyspnœa. v. Friction sound may possibly be produced by distended subpleural sacculi. vi. Muscular rumbling is sometimes heard. vii. Emphysema is often associated with pulmonary catarrh, and the physical signs are changed accordingly. Dilatation of the heart is a consequence of long-standing emphysema.

§ III. Emphysema has been confounded with pneumothorax. But bilateral pneumothorax is incompatible with life, and emphysema is bilateral. Moreover, amphoric signs are never present in emphysema.

CHAPTER VI.

PULMONARY ATROPHY.

ATROPHY of the lungs, with enlarged air-sacs, occurs as a part of general senile atrophy, or as a consequence of previous pulmonary disease, especially arrested tubercle.

§ I. There are no physical signs essentially belonging to the lesion.

§ II. Its accidental characters are these: i. Shape of chest uncertain, except that it is not bilaterally enlarged. ii. But it has all the fixed and inexpansible look of emphysema; the sterno-mastoid muscles stand out strongly against the sunken supra-clavicular spaces, unless they be bulged by a forcible expiration. iii. Diaphragm depressed; epigastric pulsation. iv. Heart covered by lung. v. Tympanitic percussion note over front of chest: the note is less resonant behind. vi. Signs of catarrh are common.

§ III. To these characters add the negative condition, that there are no definite signs of other disease; and we have all the conditions which render physical diagnosis of pulmonary atrophy possible in a patient whose symptoms point to disease of the lungs.

CHAPTER VII.

ASTHMA.

§ I. The physical signs proper to asthma; or, in other words, the signs of an asthmatic paroxysm:—i. Expiratory dyspnœa; expiration forced and greatly prolonged;[1] lower parts of chest fixed and immovable; no abdominal movements of respiration, or hardly any. This is the rule: but the signs of dyspnœa (that is to say, the powerful movements) may be more marked during inspiration than during expiration: the dyspnœa being of inspiratory type, and denoted by considerable inspiratory expansion of the upper chest, associated with great recession of the lower chest and of the root of the neck. ii. Chest bilaterally dilated during the paroxysm: diaphragm depressed. iii. Breath sounds weakened, sometimes almost inaudible. iv. Sonorous and sibilant rales common: mucous rales also towards the end of the attack. v. Heart sounds very weak, being heard through inflated lung.

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[1] Anthony Henly's Farmer dying of an *Asthma*, said, "Well, if I can get this Breath once *out*, I'll take care it shall never get *in* again." Swift's Miscellanies, vol. i. p. 263.

§ II. In the intervals of the paroxysm, the patient usually affords signs of pulmonary emphysema, atrophy, or catarrh

CHAPTER VIII.

PULMONARY COLLAPSE.

§ I. Collapse of a few vesicles is indicated when deep inspiration brings out crepitant rale, audible for a few breaths only, and then heard no more for a time : a sign common at the base of the lung, back or front.

§ II. Collapse of larger portions of lung requires, for diagnosis, two conditions, namely, that the collapse be extensive, and in contact with the chest-wall.

¶ I. Extensive collapse of this kind sometimes occurs acutely. A lesion most common in young children, and especially in children feeble or rickety, and suffering from pulmonary catarrh, hooping cough, croup, or disease of the brain. The signs are much the same as those of sudden plugging of a large bronchus (chap. ix.), and are dependent upon solidified and contracted lung. i. The base or even the whole of one side of the chest is shrunken. ii. Movements on the same side much impaired. iii. Dulness to percussion over the collapsed part: and sometimes a tracheal note in the parts around. iv. When the front of the left lung is collapsed, the heart is uncovered, and its area of percussion dulness increased. When the left lower lobe is collapsed, the heart is displaced to the left. When the right lower lobe is collapsed, the heart is displaced to the right, and the liver rises. v. Bronchial breathing over the area of dulness: or simply weak breathing if there be much mucus in the tubes. vi. Rales are usually present; and will be quite gurgling in character, if the collapse surround large tubes, such as those at the root of the lung. The diagnosis from pneumonia is sometimes impossible at first.

¶ II. Extensive collapse may be developed more gradually. i. The conditions and the signs are sometimes the same as those of the acute collapse just described. ii. In typhoid fever we sometimes meet with signs, due to simple temporary collapse, but which, when discovered in a patient examined for the first time, may be thought due to phthisis: namely, dulness to percussion at one apex, weak or puerile breathing there, and universal sonorous or mucous rales: signs which last not more than a day or two.

iii. Collapse of the right lower lobe is sometimes dependent upon great enlargement of the liver; collapse of the left lower lobe sometimes occurs in dilated heart or pericardial effusion. The diagnosis from pleural effusion depends upon the result of puncturing the chest. iv. Cirrhosis is apt to supervene upon that chronic collapse which is secondary to any form of pleurisy. Chronic collapse and cirrhosis cannot be distinguished during life; for which reason Laennec[1] called them both by the name of carnification (see chap. xxv.). The signs of both diseases are those of solidified and contracted lung. *a.* If the carnification involve only a portion of the lung (as is usually the case), this portion is the lower lobe: and the corresponding signs are, contraction of the chest, especially of its lower part; diminished resonance to percussion; bronchial or cavernous breathing; and signs of catarrh, and sometimes of dilated bronchi. The heart tends to be displaced more or less towards the contracted side. The diagnosis from pleural effusion depends chiefly upon the result of puncture. The diagnosis from tubercular phthisis depends upon the fact that the lower part of the lung is affected, upon microscopical examination of the sputa, and upon the whole history of the case.[1] β. If the carnification involve a whole lung (which is seldom the case), the affected side is shrunken and the opposite side distended, the dulness to percussion is absolute, vocal vibration weak, breathing sounds weak, whether bronchial or not; the heart becomes dilated. If the left lung be carnified, the heart will be extensively uncovered, and its pulsations widely visible. If the right lung be carnified, the heart may be so completely embedded in the same lung, as to be undiscoverable during life. Extreme shrinking of the right lung may in other cases cause the heart to lie so wholly in the right side of the chest, and uncovered by lung, that congenital malposition of the heart is simulated. The diagnosis between a lung entirely carnified and a pleural effusion depends upon puncture: in the case of a concealed or displaced heart, puncture must obviously be made with caution.

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[1] Ausc. méd. 2nd edit. vol. ii. p. 224.

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[1] Chronic Solidification of Base of Lung: St. Barthol. Hosp. Reps. vol. xxviii. p. 1: 1892.

CHAPTER IX.

PLUGGING OF TRACHEA OR BRONCHUS.

OBSTRUCTION within the trachea must obviously be very incomplete. But a main bronchus, or a large branch thereof, may be completely plugged for a considerable time: it is to this condition that the following remarks for the most part relate.

§ I. The earlier signs of a plugged bronchus are: i. Inspiratory dyspnœa, attended by imperfect movements of the chest-walls, on the affected side. Inspiratory recession of the base of the chest may involve both sides, even when one bronchus only is obstructed.[1] ii. Percussion-note not much affected at first; but it tends to lose clearness of tone if the obstruction continue and collapse ensue. iii. Respiratory sounds weakened, or even abolished according to the completeness of the plugging. iv. A whistling sound or rale (rhonchus, sibilus, stridor), inspiratory and expiratory, produced at the seat of obstruction, the plugging being incomplete. The rale is usually loud, and heard over a great extent of chest: the seat of the obstruction is not necessarily nearest to the spot where the rale is heard loudest. v. A palpable thrill, due to the same vibration as the rale: inspiratory or expiratory; felt over one side or both.

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[1] See St. Bart. Hosp. Reports, vol. xvi. p. 53: 1880.

§ II. The later signs of a plugged bronchus are due, first to simple collapse of the affected part of lung, and afterwards to destructive pneumonia. i. Permanent recession of the chest-wall is common; when unilateral, the other side is sometimes distended. Yet the shape of the chest is not always altered; it is in a few cases quite natural. Corresponding displacement of mediastinum and diaphragm. ii. Immobility of affected side. iii. Percussion tone over the affected part much impaired; it may be, to absolute dulness. iv. The auscultation signs depend greatly upon the degree of bronchial obstruction; sometimes there is little or no breathing sound; sometimes there are bronchial or cavernous breathing, and rales more or less cavernous.[1]

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[1] See chapters viii. and xvii.

CHAPTER X.

PLEURISY.

ALTHOUGH pleurisy cannot exist without inflammatory effusion, yet the term, pleurisy with effusion, is applied to those cases only which are attended by liquid effusion; these will be discussed hereafter. The present chapter relates to pleurisy attended by effusion of coagulable lymph only.

§ I. Pleurisy of this kind is often very local. The sign, not always present, is local friction sound. The resonance of the affected part of the chest may or may not be somewhat impaired.

§ II. Pleurisy of this kind is sometimes universal, involving the whole of one pleura. i. The affected side is retracted, it may be considerably, and moves much less freely than in health. ii. The percussion-note is raised in pitch and muffled, over the greater part or the whole of the side. The sense of resistence is increased. When the disease affects the left side, the superficial area of cardiac dulness is extended. iii. The respiration generally is weak, and attended by friction sound (especially in the complemental space), or by wide-spread rale, indistinguishable from the mucous rale of catarrh or phthisis. At places the breath-sound may be bronchial, in all degrees of intensity, up to perfect cavernous resonance. iv. Add to the physical signs hectic fever, and we can understand why pleurisy of this kind is often mistaken for phthisis more or less advanced. However, the pleuritic patients recover completely, without a vestige of disease left behind, save haply a slight unilateral retraction of the chest, or a cup-like depression. Whenever the signs of a case of supposed phthisis are in some respects peculiar; whenever they indicate advanced and extensive disease but limited to one side of the chest; whenever cavernous signs are heard in unusual places; it is well to weigh the possibility of simple pleurisy. As a rule, the signs of pleurisy are more marked in the lowermost part of the chest, and the signs of phthisis at the upper part.

CHAPTER XI.

PNEUMOTHORAX.

THE varieties of pneumothorax are these :—

1st. Air and liquid (pus or serum) in the cavity :	
i. Cavity large :	
α. closed . .	Hydropneumothorax.
β. with external fistula . .	Fistulous Empyema.
ii. Cavity small. .	Loculated Pneumothorax.
2nd. Air alone in the cavity	Pure Pneumothorax.

Art. I.—CLOSED HYDROPNEUMOTHORAX.

§ I. Its physical signs are these :—Unilateral distension of chest, tympanitic percussion-sound, weakened respiration and amphoric phænomena.

¶ I. Distension of the chest is indicated by :—i. Unilateral enlargement ; sometimes so great as to cause an excess of three inches in the semicircumference on the affected side. ii. Depression of the diaphragm ; sometimes so great as to force the upper surface of the liver altogether below the level of the costal margin in front, and to produce a band of tympanic resonance in the abdomen, above the liver dulness. iii. Displacement of the mediastinum towards the unaffected side occurs almost instantaneously in perforative pneumothorax ; at first due no doubt to traction exerted by the lung of the opposite side ; but before long the air in the pneumothorax comes to exert positive pressure upon the mediastinum (p. 177).

¶ II. The percussion-sound falls in pitch and increases in duration, that is to say, becomes tympanitic in proportion to the distension of the pleura. The note, however, remains muffled ; and, in fact, when the distension is extreme, the muffling approaches dulness. The metallic ring is very seldom heard unless the ear be applied to the chest (by auscultation) during percussion ; and thus heard, the metallic ring is nothing but the bell-sound. Where liquid is present, non-resonance will be found : the liquid effusion is free, movable, and changes its position with change in the position of the body.

¶ III. The respiratory sound is weakened in proportion to the collapse of the lung. Sometimes collapse is so complete, that no breathing is audible, except in the vertebral groove. When the lung has been solidified by previous disease, so that collapse cannot ensue, a respiratory sound, more or less loud and bronchial or amphoric, will be heard all over the pneumothorax.

¶ IV. Amphoric signs, indicative of a large cavity, are present. Puncture of the pleura, even when it does no more than make pressure equal within and without the chest, (the pneumothorax remaining) will sometimes remove amphoric hum and bell-sound for a time. i. Amphoric hum attends the sounds of breathing, coughing, or talking. And let it not be supposed that amphoric respiration is necessarily due to air passing out of the lung into the pleural cavity and back again: on the contrary, this is seldom the case; the breath sounds heard are pulmonary sounds, and acquire their amphoric quality by transmission through the pneumothorax. ii. Metallic tinkling may be present. iii. The bell-sound is the most constant sign of pneumothorax. iv. Succussion splash may be produced when the quantity of liquid is neither too small nor too great.

§ II. Inspiratory movement of the affected side is non-expansive: vocal thrill is diminished, or even abolished. The respiration on the unaffected side is puerile. When pneumothorax is secondary to phthisis, the apex of the lung often remains adherent. In old pyopneumothorax a large ulcerous opening is sometimes formed between the cavity and a large air-tube: thereupon, all active distension of the side ceases. In rare cases air is effused into the pericardium as well as into the pleura, whereby the signs indicative of displaced mediastinum are apt to be lost.

§ III. No disease of the chest affords signs more characteristic than those of pneumothorax, or can be discovered with greater ease and certainty, or is more often overlooked. The chief cause of this frequent failure in diagnosis is the fact that the observer is misled by the resonant percussion-note into an assumption that the affected side is natural, and so into neglect of auscultation whereby alone pneumothorax can be discovered.

Art. II.—FISTULOUS EMPYEMA:

Differs from closed hydropneumothorax in that the affected side is distended very slightly or not at all, or more frequently is contracted. Consequently the signs due to a large air-containing cavity are seldom present.

Art. III.—LOCULATED PNEUMOTHORAX:

Is most often met with at the base of one pleura. The cavity usually contains pus as well as air: whether amphoric signs be present or not, depends upon the amount of air. The signs sometimes vary with the position of the patient; thus, in the lying posture amphoric signs may be detected, which are quite absent, and replaced by the ordinary signs of empyema, in the upright posture. Pyopneumothorax of the base is often secondary to an emphysematous abscess below the diaphragm (p. 211). A loculated pneumothorax, which communicates with a large bronchus through a fistulous ulcer of the lung, can hardly be distinguished from a cavity formed within the lung.

Art. IV.—PURE PNEUMOTHORAX:

Is uncommon and mostly due to injury. The physical signs are the same as those of a closed hydropneumothorax, excepting those which depend upon the presence of liquid.

Rupture of the diaphragm, on the left side, is apt to be followed by a state of things which has been mistaken, during life, for pneumothorax.[1] The stomach and colon pass up into the pleural sac; they become greatly distended with gas; the lungcollapses. Hence displacement of the mediastinum, such that the heart beats to the right of the sternum; tympanitic percussion-note over nearly the whole of the left side; and great weakening or abolition of the breathing sounds there. The bell-sound will probably, the metallic tinkle and succussion splash may possibly, be present. Enough, in this place, to have pointed out the necessity for a cautious diagnosis.

In a case of great contraction of the left lung (due to phthisis), the distended stomach has been known to mount so high into the thorax, as to lead to the diagnosis of pneumothorax.

An abscess below the diaphragm may become partly filled with air, and may so raise the diaphragm, the pleura remaining uninjured, as to afford not only a tympanitic percussion-note over the lower part of the chest, but also some of the amphoric signs of pneumothorax. The breathing and heart sounds, conducted from the thorax, sometimes (not always) acquire amphoric quality by transmission through the air-cavity. A more constant auscultation-sign is the bell-sound. An air-containing sac between the liver and diaphragm, as contrasted with loculated pneumothorax of the right base, tends to cause much less displacement of the heart to the left, and much more displacement of the liver downwards. But the position of the liver cannot always be determined (and perhaps is not always much changed) when the air lies, for the most part, between the liver and the front part of the diaphragm and abdominal wall.[1] Subphrenic abscesses are sometimes accompanied by thoracic empyema, the diaphragm being perforated or not; if perforated, there is a pyopneumothorax also.

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[1] Laennec: Ausc. méd. vol. ii. p. 378.

Congenital deficiency of half the diaphragm produces similar results: see Polaillon, Union Médicale, no. 97: 1881.

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[1] See Coupland: Brit. Med. Journ., Mar. 23, 1889 p. 636.

CHAPTER XII.

HYDROTHORAX.

HYDROTHORAX and œdema of the lungs, pleural and pulmonary dropsy, often co-exist. Hydrothorax tends to be bilateral, although the quantity of effusion is not always equal on both sides. And, being bilateral, the fluid cannot occupy more than a portion of the pleural cavities.

i. When pulmonary œdema also is present, it is possible for great inspiratory dyspnœa, such as to simulate laryngeal obstruction, to concur with copious hydrothorax. ii. The diaphragm is depressed : the base of the thorax expanded : the position of the mediastinum remains unchanged. iii. Dulness to percussion co-extensive with the effusion. iv. Respiratory sound and vocal resonance either simply weak or feebly bronchial, over the regions where the percussion-note is dull : sometimes a little mucous rale.

CHAPTER XIII.

PLEURISY WITH EFFUSION.

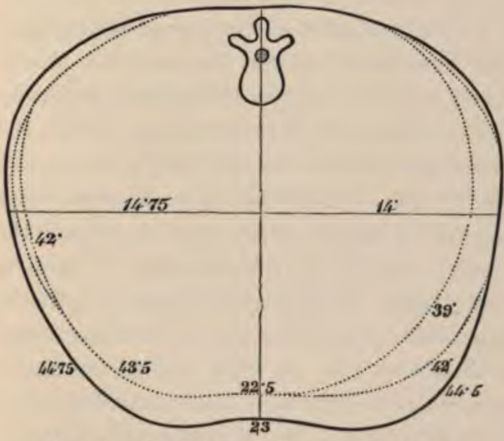
PLEURISY with effusion is usually unilateral. Its course may be divided into periods of increase, height, and decline.

§ I. Period of increase.

¶ I. The earliest sign of pleurisy going on to effusion may be either friction sound or pleuritic rale (p. 205). The friction is usually very local: its common situation being over the base of the lung, in front or at the side. The rale is more extensive, and is sometimes heard over the whole of one side of the back.

¶ II. More frequently, however, the earliest signs are those of liquid effusion: the same signs speedily supersede any friction sound or pleuritic rale which may have preceded them. The liquid tends to collect, as soon as formed, in the lowest place: what constitutes the lowest place depends upon the attitude assumed by the patient while effusion is going on. At first, when the quantity is small, the lung is simply relaxed, and swims upon the effusion: but as the liquid accumulates it compresses the lung, and renders it more or less empty of air. i. The great sign of liquid effusion is a co-extensive dulness to percussion. This dulness is not wholly due to the effusion, but is partly dependent upon associated collapse of lung; that is to say, a layer of liquid an inch or more thick would transmit percussion resonance of the lung, were the lung resonant (p. 84). Dulness begins at the lowest part of the chest behind, above the complemental space (p. 174); the note being natural elsewhere. When the effusion rises higher than the angle of the scapula, the lung will have relaxed to such an extent as to give a clear subtympanitic or tracheal note above the nipple of the same side in front: a sign not always present even in cases watched day by day from the onset. Whether, by further increase in the quantity of the fluid, the whole back becomes dull before the front is so at all, or whether the upper level of the fluid be comparatively horizontal, depends upon the attitude assumed by the patient while the effusion was going on. Hence, when the effusion is small, the dulness may be wholly posterior, and sharply defined in front by the posterior axillary line, the lateral region remaining resonant. On the other hand, the upper limit of a dulness which occupies the lower and not the hinder part of the chest, often rises higher in the axillary region than in the back. Even when absolute dulness is confined to the base, there is usually some impairment of resonance all over the back on that side. The dulness over the effusion may be far from absolute. The anterior clear resonance, when present, is sometimes of cracked-pot quality. ii. In proportion to the amount of effusion, the side is enlarged, diaphragm depressed, and mediastinum displaced. Yet even this rule is not constant: the heart may remain unmoved by an effusion of not less than a quart of serum into one pleura. iii. Vocal thrill is diminished where dulness to percussion exists, and is wholly abolished in great distension of the side. iv. The respiration is at first weakly vesicular, and sometimes remains so throughout the disease. But mostly the breathing soon becomes bronchial; sometimes even before the dulness becomes absolute. With progressive increase of effusion, the bronchial breathing tends to become less and less loud until, at last, it is wholly suppressed. But sometimes, although the quantity of fluid be very great, loud bronchial breathing is heard all over the affected side : the fact being that the loudness depends, not inversely upon the quantity of fluid effused, but directly upon the openness of the air-tubes. v. Vocal resonance weak and bronchial in much the same manner as the respiratory sound. When the effusion is partial, with clear resonance in front, the bronchophony is sometimes ægophonic about the

Fig. 15.



Course of Pleurisy with Effusion, left side.

Outer line – horizontal section, before paracentesis.
Middle line (dotted) – four days after paracentesis.
Inner line (dotted) – three weeks after paracentesis.

angle of the scapula. vi. By percussing the chest in front with two coins, and auscultating behind as for the bell-sound (p. 129), a pleural effusion will sometimes be found to transmit a clear metallic sound quite unlike that heard through healthy or solid lung.[1]

§ II. The effusion at length reaches its height. This sometimes will not be until the pleural cavity is tensely full; or the effusion may stop at any point short of that extreme. When the quantity of fluid on the left side is very great, the left half of the diaphragm is occasionally depressed to such an extent that not only can the lower margin of the spleen be felt, but even its upper margin, in fact its whole outline. At the same time, the thrusting of the heart and mediastinum into the right side of the thorax may cause the right wing also of the diaphragm to be depressed to an almost equal degree; a point ascertained by examination of the liver. Percussion, moreover, may show that the liquid reaches beyond the edge of the sternum on the other side of the chest. A small protrusion, in the lateral region, distended during expiration, receding during inspiration, and due to perforation of the pleura and intercostal space, may be met with, even in moderate serous effusion. When the effusion is partial, its position does not shift easily or at all with changes in the position of the body. The semi-circumference is sometimes actually less on the diseased than on the healthy side. Lastly, it is often by no means easy to guess at the quantity of the effusion: the physical signs will sometimes seem to indicate a large or a small effusion, and paracentesis will prove the contrary.

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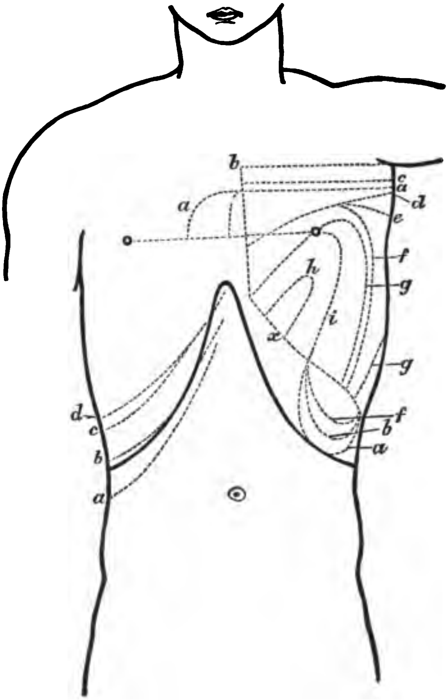
[1] Pitres; see British Med. Journ. Ap. 3, 1886. This sign was described, under the name of "signe de sou," by Sieur in a Thèse de Paris (1883), which I have not seen.

§. III. Period of decline. When a pleural effusion undergoes absorption, or is discharged by paracentesis, the following series of physical signs are noted.

The diaphragm and mediastinum go back towards their natural position: to follow the retreating organs is the best means of marking the progress of absorption, so long as the quantity of the effusion remains great. The distension of the affected side becomes less; and accurately to register this fact is an important service rendered by the cyrtometer. When the effusion has so far diminished that the lung again comes into contact with the chest-wall,

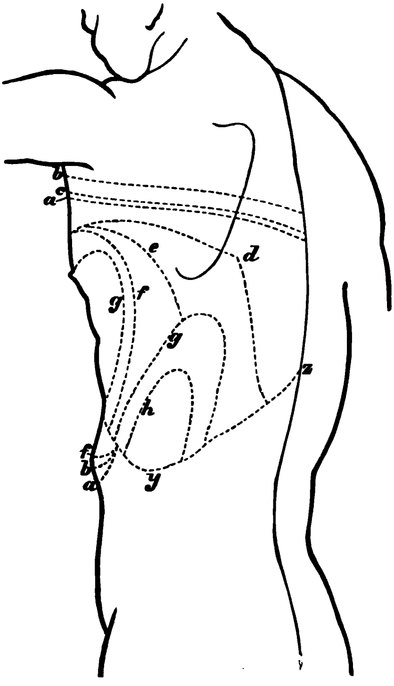
Fig. 16.



COURSE OF AN EFFUSION INTO THE LEFT PLEURA:
FROM DAMOISEAU.

a a = limits of effusion, heart, liver, and spleen, when patient first seen.
b b = same on first day of treatment.
c c = second day.
d d = third day.
e e = fourth day.
f f = fifth day.
g g = morning of sixth day.
i = evening of sixth day.
h h = last limits of effusion previous to disappearance, *i.e.*, on tenth day.
x y z = lower limit of pleura.

Fig. 17.



COURSE OF AN EFFUSION INTO THE LEFT PLEURA FROM DAMOISEAU.

percussion usually enables us to follow the falling level of fluid. And, at the same time, auscultation will sometimes inform us when and where actual contact of the opposed surfaces of the pleura has occurred; friction sound being heard.

With reference to the percussion signs more particularly. Dulness, practically absolute, and due to the unexpanded lung, often remains for a long time after perfect adhesion has occurred. The manner in which the effusion is absorbed is not constant: as a rule, the diminishing effusion follows a course more or less like that indicated in the annexed sketches.[1] The upper surface of the liquid, when it reaches as high as two inches above the nipple, is horizontal: when lower than this point, the dulness forms irregular parabolic curves, which become smaller and smaller, and last of all disappear at the lowest parts of the thorax. The fluid is mostly absorbed in the following order: from the vertebral groove near the root of the lung; from the supramammary region; from the rest of the vertebral groove and infrascapular region; from the inframammary region; and lastly from the lower lateral region; concerning which point it is important to remember that the lowest part of the pleural cavity, in the upright position, is in the axillary line. The curve of the sinking fluid is sometimes double, as happened to be the case with the patient represented in the drawing. And indeed we must be prepared to find the residue of liquid in almost any part of the chest.

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[1] Damoiseau: Arch. gén. de Méd.: Oct. 1843: p. 129.

Disappearance of effused liquid at any spot is sometimes attended, for a day or two, by friction sound, indicative of restored contact between the pleural surfaces: redux friction, as it is usually called. A large serous effusion is sometimes absorbed, not from above downwards according to the rule, but equally all over the side at once: friction or pleuritic rale becoming audible all over the side at once.

The latest physical sign, dependent upon absorption, is retraction of the affected side. Cup-like sinking of the lower part of the sternum occasionally ensues. In some cases these deformities tend to disappear gradually, in others they are permanent. The lung sometimes remains wholly unexpanded and carnified: the signs of this condition are described on p. 200.

A systolic murmur, having the characters of a pulmonary obstructive murmur, sometimes concurs with pleural effusion; disappearance of the effusion being attended by disappearance of the murmur.[1] A permanent murmur of the same kind is sometimes heard when one side of the chest is left contracted after pleurisy.

§ IV. Diagnosis. The crucial test of pleural effusion consists in puncture of the chest (p. 179). When pus is very thick, it cannot be drawn through a fine needle: in this case a larger aspirating trocar and cannula must be used. The false membranes around an old empyema are sometimes extraordinarily thick and tough: when the needle is felt to enter tissue of this kind, it must be pushed on boldly, and pus will almost certainly be reached. i. Cancer of the lung closely resembles pleural effusion in respect of physical signs. But cancer does not often cause enlargement of the affected side: nor does the dulness of cancer usually follow the laws which have been laid down with regard to pleural effusion. ii. Quickly-growing sarcoma, within a pleural cavity, has been known to distend the side of the chest, to displace the heart and diaphragm, and altogether to simulate large pleural effusion, except in the results of puncture.[2] iii. Hydatid tumours within the chest are not common: their characters will be described hereafter. iv. Chronic collapse or cirrhosis of one lung, in whole or in part, cannot be distinguished from pleural effusion, except by puncture. Collapse of the lower lobe of the left lung, dependent upon dilatation of the heart or pericardial effusion, is not easily distinguished from moderate pleural effusion, except by puncture. v. Acute pneumonia is seldom confounded with pleural effusion unless the tubes of the pneumonic lung be plugged with mucus, so that conduction of the breathing sounds is obstructed. But acute pleurisy with effusion is often wrongly supposed to be pneumonia. Much weight must be allowed to the fact that pleurisy with effusion tends to enlarge the chest, alter its shape, and displace the diaphragm and mediastinum; and that pneumonia does not. vi. But with destructive pneumonia, tubercle, and actinomycosis of the lower lobe it is different: diagnosis of these lesions from chronic loculated pleural effusion is often impossible except by puncture. And even in puncture may lurk a fallacy already alluded to, namely, that the needle may draw off a small quantity of pus from a suppurating cavity. vii. Hepatic tumours, especially hydatids and abscesses, sometimes reach so high in the chest as closely to simulate pleural effusion on the right side. The clue to diagnosis lies in the detection of hepatic enlargement by abdominal examination, and in the results of puncture. viii. To distinguish an abscess situated between the liver and the diaphragm is difficult: such an abscess will displace the heart less and the liver more than will an empyema. But both hepatic and subdiaphragmatic abscesses are often complicated with loculated empyema at the base of the right chest: in cases of this kind a trocar will sometimes pass right through the empyema and diaphragm into the abdominal abscess; the diaphragm lying high in the chest in spite of the empyema.[1] ix. Densely coagulated hæmothorax has been mistaken for empyema.[2]

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[1] Phillips: Lancet, May 25, 1889, p. 1025.

[2] De Havilland Hall: Clin. Soc. Trans. vol. xiii. p. 200.

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[1] See pp. 211 and 228.

[2] Watson: Principles and practice of physic: 4th edit. vol. ii. p. 117. 1857.

CHAPTER XIV.

EMPYEMA.

§ I. EMPYEMA of a whole pleural cavity affords the physical signs which have been described under the head of pleurisy with effusion. Often enough the distension of the affected side is anything but great: the heart for instance may be very little displaced, a fact which is sometimes due to pleural adhesion over the pericardium. The signs of pneumo-empyema (pyopneumothorax) and of fistulous empyema have been already described (p. 206).

§ II. Small collections of pus in the pleura are sometimes enclosed in dense adhesions: loculated empyemata.

¶ I. These partial empyemata occur in the following situation: i. Most commonly in the back of the pleural cavity, or between the lung and the diaphragm: the latter, or diaphragmatic empyemata, are usually larger behind than in front. ii. Less commonly in the lateral region, in the anterior region, between the lung and pericardium, or between the lobes of the lung. iii. Sometimes there are many small empyemata in one pleural cavity: sometimes there is a loculated empyema on each side.

¶ II. These partial empyemata are often complicated: when diaphragmatic, with subdiaphragmatic and hepatic abscess: when on the left side, with purulent pericarditis. Empyema on the right side of a person who has lived in the tropics, and who has suffered from dysentery or hepatitis, is often associated with deep abscess of the liver.

¶ III. The physical signs of local empyema are these. i. Chest contracted on the affected side; or contracted above and distended below; or there may be a local bulging; in the axillary region, bulging is common. ii. Breathing movements of the pleuritic side diminished. A pointing empyema sometimes moves with breathing, bulging with expiration and falling with inspiration. iii. Heart sometimes displaced, often not. Liver sometimes displaced downwards in diaphragmatic empyema: however, even in a large supradiaphragmatic empyema on the right side, the liver may not be depressed. Stomach note, when the left side is affected, often reaches high, say to the nipple level. iv. Percussion note sometimes impaired over whole of affected side, sometimes not. When the empyema is superficial, dulness at the spot: but not always absolute dulness, the tone of underlying or neighbouring lung being conducted by the effusion. Indeed over a very small empyema, there is sometimes no dulness at all. The dulness, not being due to the pleural effusion simply (p. 84), affords no measure of the extent and capacity of the empyema: dulness, absolute and almost universal, may be associated with a small empyema, the signs being chiefly due to collapsed and adherent lung. v. Respiration usually simply weak all over the affected side: sometimes bronchial where the empyema is superficial. Crackling rales are common.

¶ IV. The diagnosis has been discussed in the last chapter. When empyema is complicated with catarrh, the resemblance to tuberculosis, local or general, is great.

§ III. Pulsating Empyema: empyema which pulsates rhythmically with the heart.[1]

¶ I. The empyema is commonly large, occupies and fills the left pleural cavity. i. In a few cases, the pleural effusion (usually purulent, seldom serous) nowhere points or bulges through the chest wall. The pulsations are sometimes seen and felt over almost the whole of the side, but mostly they are limited to the normal heart region (that is to say, on the left of the sternum), or to the lowest three or four left interspaces. ii. More commonly the empyema points in one or two places, which alone pulsate. This bulging occurs in the normal heart region, or in the lowest interspaces: twice has the protrusion been seen in the loin below the ribs. The bulging is never larger than an orange.

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[1] Comby: De l'empyème pulsatile: Paris, 1882.

¶ II. In all cases of this kind, whether bulging or not, the heart is much displaced to the right: pericarditis may concur, but usually the heart is healthy. Auscultation of the pulsating part may detect conducted heart sounds. Palpation detects no thrill and no expansion like that of aneurysm.

¶ III. Paracentesis greatly helps the diagnosis. By removing part of the liquid the pulsation ceases: but the heart does not return to its natural position, being fixed by external pericardial adhesions.

¶ IV. The effusion is mostly chronic, and the lung wholly collapsed. Pneumothorax sometimes concurs : in this case the pulsation is conveyed by the liquid only.

¶ V. The diagnosis is from intrathoracic aneurysm, and from the very uncommon condition of a pulsating cancerous tumour. Aortic aneurysm and pulsating empyema may concur.

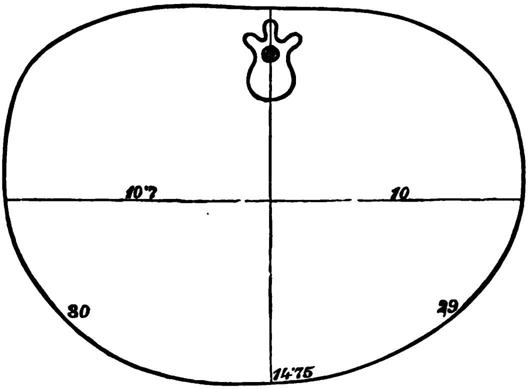
¶ VI. Very seldom the empyema does not fill the whole pleural cavity but is loculated and enclosed in adhesions. This kind of pulsating empyema always bulges, it may be to the right of the sternum, but still in close neighbourhood to the heart.

CHAPTER XV.

ADHERENT PLEURA.

§ I. VERY densely adherent pleuræ are attended by the signs of unilaterally contracted chest. The percussion sound will be impaired, and the breath sound weakened, over a large part of the affected side. Sometimes

Fig. 18.



UNILATERAL RETRACTION OF LEFT SIDE OF CHEST CONSEQUENT UPON AN ADHERENT PLEURA.

signs of chronic collapse or of cirrhosis of lung are present.

§ II. But much looser adhesions, such as are so often unexpectedly found post mortem, and which influence the percussion and auscultation of the chest in no respect, may be sometimes discovered by the cyrtometer, when one pleura only is obliterated (fig. 18).

§ III. Recovery from pleural effusion or empyema is sometimes complete, so far as physical signs are concerned ; the most careful examination failing to discover contraction of the chest, or any other sign of past disease.

CHAPTER LXVI.

PNEUMONIA.

ACUTE pneumonia is of two kinds, lobar and lobular; the solidification being, in the former case massive, and in the latter disseminated.

ART. I.—LOBAR PNEUMONIA.

§ I. The earliest distinctive signs are those which depend upon hepatisation. Of the engorgement, which precedes hepatisation, there are no constant signs. Crepitation certainly is no such sign; for it is a rale which may appear at any period of the disease, and which in many cases does not appear at all: seldom are consolidation-signs preceded by crepitation; and they do not always follow it. Other precursors of the distinctive signs are met with now and then; mucous rales, and well-marked pleural friction.

The signs of hepatisation sometimes appear quite late in the disease, for instance on the fifth or sixth day of fever, or even not until defervescence. Indeed there may be no physical signs whatever in the course of a disease which is undoubtedly pneumonia.

§ II. Hepatisation of lung is characterised by dulness to percussion, bronchial breathing, and bronchophony. i. In persons whose chest-walls are resilient, the dulness is less absolute than that afforded by pleural liquid effusion, cancer of the lung, or dense tubercular consolidation. A muffled tubular note sometimes best expresses the character of the percussion sound. Clear tubular percussion note sometimes precedes the hepatisation dulness. When islets of unsolidified lung are imbedded in the surface of hepatised tissue, they afford clear tubular resonance, or even a cracked-pot sound. A cavity, or large bronchus, separated from the surface by a thin layer of pneumonic lung, will produce the same effects. ii. The bronchial breathing of pneumonia sometimes differs from that commonly met with in other diseases by being more whiffing, tubular in short. However, even in genuine lobar pneumonia, the bronchial breathing is not seldom of the ordinary softer kind. The bronchophony is mostly of a sniffing kind. All breathing sound and vocal resonance may be absent over hepatised lung: this condition is either fleeting or lasting; when fleeting, it is due to obstruction of the larger tubes by mucus, which a cough can remove; when lasting, the air-passages probably contain solid exudation or coagula. In infants the bronchophonic cry is often the only auscultation-sign which can be obtained. iii. Nigh unto the area of bronchial breathing and dulness, true crepitation is sometimes heard, over lung whose percussion tone is good, and which may or may not afterwards become dull. Reverberating mucous rales are sometimes present. Friction sounds also. iv. In lobar pneumonia of the lower half of a lung, the chest, on that side, is expanded to the state of deep inspiration; but this expansion is less than that of pleural effusion; moreover, pneumonia never displaces the heart or diaphragm. v. Pneumonia of the upper part of the lung is sometimes attended by clear tracheal percussion note in front on the same side. vi. Hepatised lung is sometimes felt to throb rhythmically with the heart. Laennec[1] believed that the solid lung simply conducted the heart's movements:

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[1] Auscult. médiate: 2nd edit. vol. ii. p. 388.

Graves[1] held that the dilated vessels of the engorged lung pulsate.

§ III. Hepatisation mostly undergoes resolution; which is characterised by progressive diminution in the bronchial quality of the breathing, by the occurrence of mucous rales and by gradual restitution of resonant percussion-note. Abundant true crepitation may be heard during this stage. The percussion-note may become tracheal and clear for a day or two. Much impaired resonance often remains for a long time after all the acuteness of the pneumonia has passed away. Occasionally the solidified tissue softens rapidly down into an abscess, a condition which does not admit of diagnosis. Unresolved pneumonia passes into cirrhosis. Pleural effusion (especially empyema) is a sequel not uncommon.

§ IV. The diagnosis of lobar pneumonia mainly relates to acute pleurisy with effusion. When breathing sounds are not heard over hepatised lung, a pleural effusion is somewhat simulated. Much more often is an acute pleural effusion, not very large, supposed to be pneumonia. In either case, if the diagnosis cannot otherwise be attained, a puncture should be made. The two diseases sometimes concur. Collapse of lung (p. 199) and acute solid œdema (p. 190) may simulate pneumonia.

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[1] Clinical lectures: 2nd edit. vol. ii. p. 39.

Art. II.—LOBULAR PNEUMONIA.

Severe catarrhal or diphtheritic bronchitis occurring in children, old people, or debilitated persons, is very apt to be accompanied by lobular pneumonia. If the pneumonic foci be discrete, the signs are not more than those due to the catarrh. If the foci be confluent over a considerable extent of lung, dulness to percussion, bronchial breathing, and sharp reverberating rales may be heard: over the middle lobe of the right lung for instance.

CHAPTER XVII.

DESTRUCTIVE PNEUMONIA AND PULMONARY GANGRENE.

DESTRUCTIVE pneumonia due to plugging of a bronchus has been already described (p. 203). The same lesion may be produced by compression of a bronchus by a tumour of some kind, usually aneurysmal or cancerous. The primitive pneumonic consolidation tends to ulceration and excavation, or to gangrene. Dilatation of the tubes is common.

The signs relating to the affected lung are: dulness to percussion; and, at first, weakness in any degree up to total loss, of vocal thrill, vocal resonance, and breathing sounds. Later on, signs of excavation, altogether like those of phthisis, may appear; bronchial breathing, bronchophony, and gurgling rales. The diagnosis from pleural effusion depends chiefly upon the result of puncture. Tubercular phthisis is to be distinguished by microscopic examination of the sputa.

The diagnosis of Pulmonary Gangrene, whatever be its cause, depends chiefly upon the characters of the sputa, namely, the fœtor, and the pulmonary elastic tissue to be seen by microscopic examination. When the patch of gangrene is large and single, the signs are the same as those just narrated: when the gangrenous spots are many, small, and scattered through the lung (a result of embolism), the auscultation and percussion-signs are those of diffused catarrh. The diagnosis is from other diseases attended by stinking expectoration, namely: from local empyema (by puncture); from phthisis (by microscopical examination of sputa); from dilated bronchi, with or without surrounding consolidation of lung; from mere bronchitis; and from abscess of the bronchial glands, which opens into the air-tubes.

CHAPTER XVIII.

EMBOLIC PNEUMONIA, OR PYÆMIC INFARCTUS.

¶ I. The physical signs are chiefly due to attendent pleurisy, namely: friction sound over any part of the chest; or signs of pleural effusion at the base, dulness to percussion and bronchial breathing.[1]

¶ II. The solidification itself may be large enough to afford signs. A superficial infarctus the size of a walnut, will yield, in a lean adult or a child, distinct dulness to percussion and bronchial breathing. Innumerable small scattered lesions, whether solid or purulent; yield the signs of bronchitis and nothing more.

¶ III. Pneumothorax may follow the bursting of a pyæmic abscess.

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[1] Plugging of a large pulmonary artery, by a dislocated venous thrombus, is followed by signs of œdema in the part of lung affected, when the patient has recovered from the immediate shock of the embolism.

CHAPTER XIX.

PULMONARY TUBERCULOSIS.

ACUTE or chronic pulmonary tuberculosis, if scattered, that is to say, not going on to massive consolidation or phthisis, seldom affords physical signs which have any direct relation to the tubercle.

¶ I. Latent. The lesion is sometimes wholly latent; a most copious eruption of miliary tubercle being found after death in lungs which during life yielded no physical signs of disease.

¶ II. Bronchitic. Another form of the lesion is bronchitic, the tubercles being either miliary or crude; and the signs, diffused over both lungs, are these: Percussion-note either unaltered, or somewhat raised in pitch, the resistence being increased at the same time. Breath sounds weak; sometimes faintly bronchial here and there. Mucous rales, which are sometimes sharp and reverberating, sometimes not: now and then, fine inspiratory crepitation. Signs of a more massive consolidation of a portion of the lung are sometimes present. Indeed there are all degrees between the bronchitic form of tuberculosis and common phthisis.

¶ III. Pneumonic. Acute pulmonary tuberculosis sometimes takes on a pneumonic form ; that is to say, massive tubercular consolidation occurs so rapidly as to afford crepitant rale, and most of the signs of lobar pneumonia.

CHAPTER XX.

PULMONARY PHTHISIS.

BY pulmonary phthisis is meant massive tubercular consolidation which tends to ulceration.

§ I. The physical signs of phthisis depend upon these particulars: the consolidation itself; diminished bulk of the solidified part; the localisation of rales in the solidified part; and the formation of cavities.

¶ I. i. Consolidation is indicated by diminution of percussion resonance; the pitch of the note rises and its clearness diminishes until, in some cases, absolute dulness is reached. ii. In the earlier stage of progressive solidification the respiratory sound is simply weak (an important sign); later on, it becomes more and more bronchial. The bronchial breathing, which at first is due to consolidation of the spongy structure, afterwards becomes intensified (or cavernous) by the formation of cavities. In rare cases, when the solidification is very dense and massive and not yet excavated, the respiration cannot be heard at all.

¶ II. Diminution in the bulk of the solidified part occurs early in the disease, and is attended by contraction of the corresponding region of the chest. When the left upper lobe is affected, the superficial area of cardiac dulness will be increased. That form of phthisis which ends in cirrhosis, affords the most marked shrinking of the chest: in these cases displacement of the mediastinum and of the heart, and elevation of the diaphragm occur.

¶ III. Fixed localisation of mucous (crackling, bubbling, subcrepitant) rales in a limited portion of lung, is an early and important sign of phthisis. At first more or less obscure in proportion to the weakening of the respiration, the rales gradually become clear and reverberating (or cavernous) as consolidation and ulceration proceed.

¶ IV. The diagnosis of cavity demands that it be near the surface, not smaller than a walnut, and containing fcr the most part air. i. Under these conditions the formation of a cavity is sometimes attended by a change of the previously dull percussion sound into a clear tone. When this is the case, the pitch of the tone may vary according to the openness of the patient's mouth and to his posture; but these are niceties of little or no importance. ii. A large cavity is sometimes attended by bulging of that part of the chest-wall which had previously been sunken. iii. The bronchial quality of the respiratory sound is always well marked in an empty cavity: the rales are large and resonating (cavernous). iv. The veiled puff is believed by some to be a sign of cavity. v. Practically the physical diagnosis of excavation mostly comes to this, that, in progressive phthisis, a cavity is presumed to be present where the bronchial breathing is most intense. vi. Very large cavities may afford amphoric percussion-note, amphoric hum, metallic tinkling and splashing; but seldom a bell sound. Indeed, amphoric signs are uncommon in phthisis: and yet a phthisical cavity may be so completely cleaned out and distended with air, as to yield all the amphoric sounds, to displace the heart greatly, and to simulate pneumothorax very closely.

§ II. i. The chest of persons predisposed to phthisis is usually phthinoid; and that of persons actually phthisical, flat. Inasmuch as phthisis seldom involves both lungs to an equal extent, unilateral retraction of the chest is mostly present. A much more local shrinking commonly occurs where the phthisical processes are most advanced. Occasionally the thorax is of emphysematous shape: this is the case when phthisis is engrafted upon emphysema, or when emphysema follows retrograde phthisis. ii. Cracked-pot sound is sometimes begotten by percussion over phthisical consolidation, both when a cavity is present and when it is not. Clear tracheal resonance sometimes attends incipient phthisis, when there is no reason to suspect cavity. iii. Sounds other than obvious rales, creaking and rubbing sounds, are not uncommon, and are most likely produced in the solidified tissues themselves; sometimes, however, undoubted transitory friction is heard. Collapse crepitation may be heard over phthisical lung. iv. The coexistence of a diffused pulmonary catarrh, or of emphysema, or of laryngeal disease, is a great impediment to the physical diagnosis of phthisis. Generally speaking, the physician should examine a patient, supposed to be a phthisical, several times before giving a positive opinion. v. Aneurysmal dilatation of a branch of the pulmonary artery, contained in a phthisical cavity, has been known to give rise to shrill systolic murmur audible in the suprascapular fossa.[1] Murmurs produced in the subclavian artery are not uncommon. vi. The absence of physical signs of disease, in some cases of slowly progressive pulmonary consumption, ending in death, is most remarkable. vii. The respiration in the unaffected parts of the lungs is puerile, provided it be not modified by the presence of catarrh. The loudness of the puerile breathing sometimes leads the inexpert to predicate disease just in that one part where the lung remains healthy.

§ III. The diagnosis between phthisis and some forms of catarrh, pleurisy, pneumonia, pulmonary cancer, hydatid, cirrhosis, and mycosis, is discussed in the chapters which deal with those diseases.[2]

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[1] See p. 157 for the pulsatile sounds of cavity: and note on p. 157 for pulse-breath.

[2] Pneumoconiosis (dust disease of the lung), if not tubercular, cannot be distinguished from phthisis by the physical signs alone. If syphilis, in rare cases, affect the lungs, a clinical history of the disease remains to be written.

CHAPTER XXI

PULMONARY CANCER.

¶ I. WHAT has been said of pneumonia and tubercle, is true of cancer; that small disseminated consolidations cannot be discovered by physical examination.

¶ II. The signs of massive cancer are, in general, absolute dulness to percussion, diminished or absent vocal thrill, immobility of the chest on the affected side, and weak or absent breath-sound. When a large open bronchus is intimately connected with the cancerous mass, bronchial breathing will be heard.

¶ III. Cancer sometimes causes remarkable contraction of a whole lung: in such cases, pleural effusion almost necessarily follows. The physical signs are those of the effusion: but the diagnosis can sometimes be made by the discovery of cancer elsewhere, and, in particular, of large hard glands above the collar bone (on one or both sides) or in the armpit.

¶ IV. i. Phthisis is simulated by cancer of the apex of one lung, especially when it breaks down into cavities.[1] Physical diagnosis in such a case becomes possible only when the cancer involves the mediastinum, or spreads in a manner unlike the phthisical process ; or affects other remote organs. ii. It has been already pointed out (p. 224) that very large cancerous tumours of the lung afford signs which for the most part closely resemble those of pleurisy with effusion. But cancer, unless of quickest growth, does not enlarge the chest, and may even cause it to be contracted. The mediastinum and diaphragm are seldom displaced. Nevertheless, a quickly-growing tumour sometimes sprouts so as to displace the heart or depress the diaphragm. When dulness begins not at the bottom of the chest ; when there is a great extent of absolute dulness in front, and none behind ; when, in the midst of a great extent of dulness, we detect one or more small insulated patches of resonance (haply quite clear or even cracked-pot) ; we may debate the existence of solid tumour. The crucial test is puncture. iii. Mediastinal cancer (chap. 42), and pericardial or pleural effusions often concur with pulmonary cancer. iv. Cancerous tumour of the lung, near to the heart or aorta, may (like mediastinal tumour) pulsate, and also yield a systolic murmur, it is said.[1] In such a case, the diagnosis from aortic aneurysm might be very difficult.

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[1] J. R. Bennett : Brit. Med. Journal. 1870, pp. 565 sqq.

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[1] Stokes: Diseases of the chest: 1837: p. 378. Cancer of upper lobe of left lung, causing pulsation and murmur behind manubrium sterni and its neighbourhood: the pulsation probably not in vessels of new-growth, but conducted from pulmonary artery.

CHAPTER XXII.

PULMONARY HYDATIDS.

¶ I. A HYDATID cyst may be buried so deeply within the lung as to be altogether beyond the reach of physical examination.

¶ II. A moderately large cyst is indicated by an area of percussion dulness not smaller than the palm of the hand, always rounded in shape, with a sharp outline, and situated most commonly in the lateral or infraclavicular regions.[1] Over this dull space, vocal thrill and breathing sounds are absent. Beyond the dull space, both percussion and breathing sounds are natural: portions of lung may be relaxed, just as in pleural effusion, so as to yield a percussion resonance unnaturally clear. Local hemispherical bulging sometimes occurs. Local friction also.

¶ III. A very large hydatid cyst will yield most of the signs of partial or total pleural effusion, namely, unilateral distension of the chest, displacement of the diaphragm and mediastinum, dulness to percussion, and weak or absent breathing sound.

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[1] S. Dougan Bird: already cited (p. 181).

Puncture of the chest is the most decisive means of diagnosis. The method has already been described (p. 181). Hydatid liquid escapes; unless the cyst have suppurated, in which case the diagnosis from empyema depends upon detection of echinococcus hooklets or hydatid membrane.

¶ IV. The cyst often bursts into the air-tubes: hydatid fluid, hydatid membrane, blood or pus being expectorated. A large suppurating cyst, with a free opening in the bronchia, simulates pulmonary phthisis.[1]

¶ V. The cyst has been known to burst into both the bronchia and the pleura, and so to cause a hydropneumothorax.[2]

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[1] Greenfield: Clin. Soc. Trans. vol. x. p. 103. 1877.

[2] Case quoted from Mercier by Trousseau: Clinique Médicale, vol. i. p. 711. 2nd edit. 1865.

CHAPTER XXV.

PULMONARY CIRRHOSIS.

CIRRHOSIS, sclerosis, or grey induration commonly affects a portion only (and that usually the base) of one lung; seldom affects a whole lung; and both lungs more seldom still.

¶ I. The physical signs depend upon the solidification and shrinking of the lung which cirrhosis causes. i. The solidification signs are:—percussion dulness more or less absolute: breathing sounds either simply weak, or bronchial in any degree up to most highly marked cavernous breathing: vocal resonance unaffected, or simply weakened, or bronchophonic. ii. The signs of unilateral or local shrinking have been already described in several places (pp. 29, 32, 200, 201).

¶ II. Other lesions are apt to complicate cirrhosis. i. Catarrh, with its attendent rales in the affected part. ii. Dilatation of air-tubes affording signs discussed in the foregoing chapter. iii. Adhesion of the pleural surfaces by a thick and tight membrane : almost certainly present when unilateral contraction of the chest is great. iv. In very chronic cases ; emphysema of the other lung, and dilatation of the heart.

¶ III. Diagnosis from tubercular phthisis cannot be attained by physical examination alone : chronic consolidation of the base of a lung is more likely to be cirrhotic than tubercular.[1] Any question of pleural effusion must be determined by puncture. Simple chronic collapse (whether dependent upon past pleurisy or not) cannot be distinguished from cirrhosis : Laennec included both lesions under the name of carnification : the physical signs have been fully described in the chapter on pulmonary collapse (p. 200).

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[1] St. Barthol. Hosp. Reps. vol. xxviii. p. 1.

CHAPTER XXVI.

HYPERTROPHY OF THE HEART.

THE heart is said to be hypertrophied when the quantity of its muscular tissue is increased. The sign is a heaving impulse, which is best felt at or just above the apex-beat when hypertrophy affects the left ventricle ; and a little to the left of the lower part of the sternum when hypertrophy affects the right ventricle ; of hypertrophy of the auricles there is no certain sign. The heaving impulse may be countervailed or concealed by conditions such as these ; feeble action of the heart, degeneration of its texture, emphysematous lung over the heart, and very fat chest-walls : wherefore heaving impulse is far from being a constant sign of hypertrophy. When the right ventricle is hypertrophied, the conducted epigastric impulse is strong. When the left ventricle is hypertrophied, the apex of the heart sometimes reaches farther to the left than natural, partly perhaps in consequence of elongation of the aorta associated with the hypertrophy.

A sharp whizz is heard in the arteries under a certain degree of pressure by the stethoscope; and the pulsation may be strong enough to raise the observer's head at the same time (p. 161).

CHAPTER XXVII.

DILATATION OF THE HEART.

¶ I. The signs of dilated heart are these: i. Bulging of the heart region (p. 31). ii. Extension of the deep-seated area of cardiac percussion dulness, along a horizontal line; to the right when the right cavities, and to the left when the left cavities are dilated. The dull space usually remains oval in shape; a point upon which the diagnosis between an enlarged heart and a pericardial effusion greatly depends. Yet the deep dulness of dilated heart may be sometimes extended upwards so as to reach the first left cartilage, and to assume a shape more or less triangular. iii. The apex-beat moves with the dilatation and extended dulness to the left; they all sometimes reach to the left as far as the axillary line. iv. An enlarged heart tends to displace the lungs, and so to come into contact with a larger space of the chest-wall; hence an impulse more extensive than natural.

¶ II. The diaphragm is depressed; sometimes, but seldom, so much that a liver of

Fig. 19.

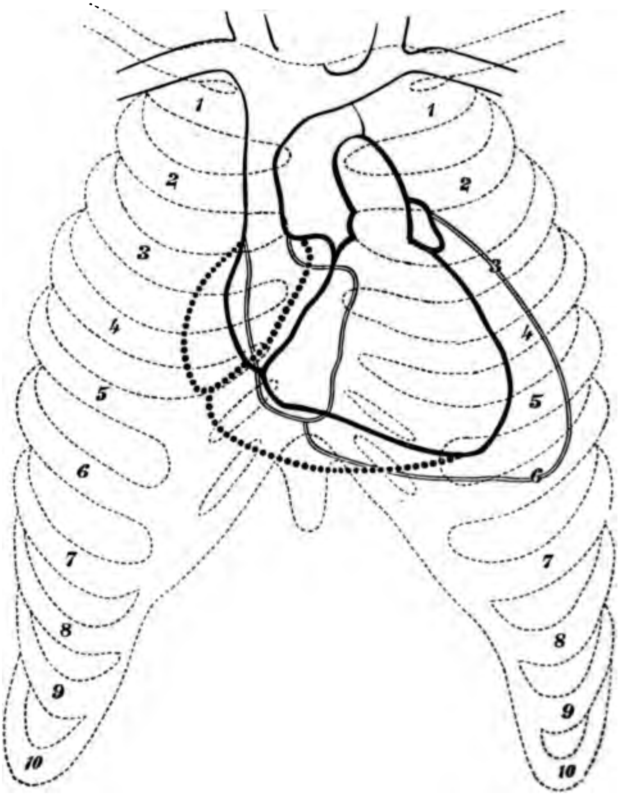

DIAGRAM SHOWING DILATATION OF THE RIGHT SIDE AND OF THE LEFT SIDE OF THE HEART: FROM VON DUSCH.

natural size may seem to be enlarged. Dilatation of the right cavities is usually accompanied by over-filling and pulsation of the jugular veins (p. 166). A dilated right auricle has been known to produce a systolic impulse in the fifth right interspace, two inches from the sternum (p. 42). Chronic collapse of the lower lobe of the left lung is sometimes caused by dilated heart (p. 200).

¶ III. Dilatation of the heart may be simulated by pericardial effusion; by intrathoracic tumour (aneurysmal or not) above the base of the heart, or pushing the heart forwards against the front of the chest; by mere increased extent of contact between the heart and the chest-wall, dependent upon shrinking of the left lung; by consolidation of the anterior part of either lung; and by local pleural effusion. But difficulty in the diagnosis seldom arises except in the case of pericardial effusion (p. 268). Dilatation of the heart is usually associated with signs of valvular disease. The apex-beat may be displaced to the left, not only by dilatation of the left ventricle, but also by dilatation of the right heart alone, by elongation of the aorta, displacement of the mediastinum to the left, and elevation of the diaphragm (p. 41).

CHAPTER XXVIII.

PERICARDITIS.

THE physical signs of pericarditis depend upon inflammatory effusion into the pericardium. When the quantity of exudation is small, it cannot be discovered, unless it cause a friction-sound (p. 153). For this reason, a friction-sound is usually the earliest sign of pericarditis. When the quantity of exudation is large, it is discovered by means of percussion (p. 265). Disappearance of friction-sound is due either to progressing liquid effusion, or to adhesion of the inflamed surfaces, or perhaps to absorption of exudation, or to diminution of hyperæmia. But friction is not always abolished even by large liquid effusion. On the other hand, there will sometimes be no friction, even when the pericardium is full of coagulable lymph with very little serum. And lastly, pericarditis is sometimes wholly undiscoverable either by percussion or by auscultation: the lymph may be too soft to yield friction-sound and a liquid effusion (even of some ounces) may lie behind the heart so as not to affect the area of its percussion dulness.

It is sometimes hard to decide whether friction-sound, heard over the heart region, is pleural or pericardial. Pleural friction may be developed by movement of the heart alone, and pericardial friction may be under the influence of breathing movements (pp. 127, 154, 158).

CHAPTER XXIX.

PERICARDIAL EFFUSION.

INCREASE in the area of cardiac dulness is the sign, not only of dilated heart, but also of effusion (of serum, pus, or soft and bulky lymph) into the pericardium. The two diseases, however, do not extend the area of dulness in the same manner ; hence the means of diagnosis between them. Dilatation of the heart has been already spoken of : it remains to describe the signs of pericardial effusion.

§ I. The first extension of percussion dulness occurs at the base of the heart, where the great vessels enter, and where the pericardium hangs loosely round them, and is most distensible. At the beginning, the dulness is increased chiefly upwards, so as to reach the second left rib, between the sternum and the parasternal line. A larger effusion will extend the dulness at the base transversely, so as to reach from the right side-sternal, or parasternal, line to the left nipple line, and as high, it may be, as the first rib.

Hitherto there will have been little change in the signs afforded by that part of the pericardium which is close upon the diaphragm. However, further increase of effusion, after it has distended the pericardial sac around the vessels, will dilate the pericardial sac around the heart. Hence, progressive increase in transverse dulness below the base of the heart, and corresponding displacement of the lungs, until, in an extreme effusion, the non-resonant space will reach from the right nipple line to the left axillary line, and up to the top of manubrium sterni, the whole left front being dull. In all pericardial effusions, whether great or small, it is upward extension of dulness which affords the chief means of diagnosis from enlargement of the heart.

§ II. Pericardial effusion may, as already said, lie wholly behind the heart, and be undiscoverable, even when there are no old adhesions in the sac to account for this peculiarity. Lung, emphysematous or not, and air-distended, lying in front of the pericardium, will obviously interfere with the development of percussion dulness, such as just described. Dulness behind the manubrium sterni is sometimes not present, even although the pericardium, distended with liquid and uncovered by lung, lie there: the reason being that the liquid conducts a clear note from the trachea or neighbouring lung. Acute dilatation of the heart's cavities sometimes concurs with pericarditis, and will, when associated with small effusion around the vessels at the base of the heart, give physical signs of large effusion. Chronic dilatation of the heart will produce the same effect, when the pericardial sac is everywhere obliterated by old adhesions, except at the base around the large vessels, and effusion takes place there.

§ III. When effusion is limited to the base of the heart, the apex-beat and left limit of dulness will correspond ; but when the cardiac portion of the pericardium is distended by liquid, the dulness will reach beyond and to the left of the apex-beat. At the same time the impulse is weakened ; it may finally become imperceptible even when the patient is lying upon his face. An impulse, wholly impalpable in the supine posture, may be well felt in the erect. Bulging of the heart region is consequent upon large pericardial effusion as well as upon dilated heart, especially in the young. In a few cases of excessive distension, a sort of undulation has been seen, comparable with pulsating empyema. The heart sounds are weakened more or less, and may become almost or quite inaudible : a condition which must be due to weak action of the heart as well as to the effusion.

§ IV. The lung around a distended pericardium is relaxed. A large effusion sometimes exerts so much pressure upon the bronchial tubes, especially the left, as to cause more or less extensive collapse of the corresponding lung (pp. 200, 262). Great bulging of the left lung-apex above the clavicle, dependent upon pericardial effusion, has been noted.[1] The cervical veins are sometimes very full. The diaphragm is depressed, and therewith the liver and the spleen :[2] seldom is the depression so great as to produce swelling of the epigastrium.[3] The diaphragm may be paralysed ; if it be, the epigastrium sinks inwards during inspiration.

§ V. The diagnosis is from dilatation of the heart ; aneurysms, abscesses, or other mediastinal tumours ; consolidation of the front parts of the lung ; and pleural effusion. i. From dilatation of the heart the diagnosis depends mainly upon the different shapes of the area of percussion dulness in the two diseases. Yet it cannot be denied that a dilated heart sometimes yields that upward extension of dulness which is the chief note of pericardial effusion: hence the instances, not very few, of the heart having been pierced in an attempt to draw off a non-existent pericardial effusion. On the other hand, much horizontal extension of percussion dulness to the right of the sternum, in cases of pericardial effusion, will sometimes lead to the mistaken diagnosis of dilated heart. So that the whole history and all the physical signs must be pondered in a difficult case. The combination of dilated heart and pericardial effusion has been already spoken of (p. 267). Puncture of the pericardium is never performed as a mere means of diagnosis (p. 180). ii. From aneurysmal and mediastinal tumours the diagnosis depends upon careful comparison of all the physical signs of the two diseases. Pericardial effusion seldom affords signs of pressure upon the innominate veins or recurrent laryngeal nerve. iii. From consolidation of lung the diagnosis depends mainly upon the auscultation signs. iv. Loculated pleural effusion, situated exactly over the heart and only there, seldom occurs. Large effusion into the left pleura may cause bulging of the chest in the heart region, such as to raise the question of a concurrent pericardial effusion: for pleurisy on the left side and pericarditis are often associated. The diagnosis depends mainly upon the result of paracentesis, and emptying the left pleura, whereby alone can the signs of pericardial effusion become manifest.

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[1] Graves : Clinical Lectures, 2nd edit. vol. ii. p. 176.

[2] Senac : Traité, 2nd edit. vol. ii. p. 364. 1783.

[3] Auenbrugger's Inventum Novum, § 46.

CHAPTER XXX.

PNEUMOPERICARDIUM.

THE percussion note, over the heart region, is clear (and sometimes attended by a metallic ring) when the patient is lying : when he sits up or leans forward, the sound becomes dull. A cracked-pot sound has been noted.

The auscultation signs are these :

i. Amphoric quality of the heart sounds.

ii. Friction sound, if present, acquires the character of metallic tinkling.[1]

iii. When the pericardium contains liquid as well as air, the water-wheel sound (p. 155) is heard. It is sometimes so loud as to hide the heart sounds, or even to be audible without auscultation.

iv. Succussion splash has been heard in cases of the same kind.

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[1] Graves : Clinical medicine, 1843 : p. 824. Hepatic abscess which opened into both stomach and pericardium.

CHAPTER XXXI.

ADHERENT PERICARDIUM.

THERE are no certain signs of adherent pericardium. When the pericardium adheres closely, not only to the heart within, but also (by means of comitant pleural adhesion) to the walls of the chest in front and to the spinal column behind, the following signs may be present. i. Systolic recession of the apex-beat, associated with a distinct impulse : a sign which even if well marked cannot be wholly depended upon (p. 43). ii. Deep inspiration does not diminish the area of superficial cardiac dulness ; does not depress the apex-beat ; and is attended by recession of the epigastrium,[1] consequent upon fixedness of the pericardial portion of the diaphragm : but these are signs rather of external than of internal pericardial adhesion : however pericardial and neighbouring pleural adhesions often concur. In universal tough pericardial adhesion, it is common enough that the signs are no more than those of attendent dilatation of the heart and its cuspid orifices.

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[1] See Laennec : vol. ii. p. 665.

CHAPTER XXXII.

MITRAL REGURGITATION.

¶ I. A MURMUR, replacing or immediately following the first sound, and heard louder at or just above the apex-beat than over any other part of the heart region, indicates regurgitation through the mitral orifice.[1] The murmur is often heard well at the angle of the left scapula: is sometimes louder there than at the apex-beat; is sometimes indeed heard at the angle of the scapula only, and not at all at the apex-beat. The murmur is often heard well, or even heard loudest, over the second left intercostal space or third left cartilage, at one or two inches from the left edge of the sternum: behind which spot lies the tip of the left auricular appendix, whereinto the murmur is supposed to be conveyed by regurgitation. The murmur is sometimes heard over a very small space at the apex-beat, and nowhere else. On the other hand, the murmur is sometimes conveyed far away from the heart, so as to be audible over the head or sacrum, for example.

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[1] "The opinion of the permanent patency of a cardiac opening from any cause, as a source of the bellows-sound, I heard first from Dr. James Johnson. Who originally suspected it I cannot say." Elliotson: On the recent improvements, etc., 1830: p. 20. Hope claims the discovery for himself: Treatise, 4th edit. p. xxvi. and p. 70.

¶ II. Incompetence of the mitral valve is not always attended by a definite murmur: this is especially the case when the heart is much dilated and unable to contract duly upon its contents: wherefore mitral murmur often disappears before death. Moreover, under any circumstances, mitral murmurs are of all murmurs most variable: they disappear and reappear in an inexplicable manner. They are sometimes affected by breathing, being weakened or even becoming inaudible, during inspiration: and this when no question arises of pulsatile respiratory murmur (p. 156). Systolic apex thrill (p. 53) and accentuation of the pulmonary second sound (p. 137) are often to be noted.

¶ III. Mitral regurgitation is usually due to disease of the valvular cusps. But not always so: no doubt the orifice is sometimes simply dilated, partaking in primary dilatation of the heart (not secondary to valvular disease): in which case the relative shortening of the musculi papillares and chordæ tendineæ will increase the incompetency of the cusps. Moreover, it is possible that debility or degeneration of the musculi papillares or of the muscular sphincter which surrounds the mitral orifice,[1] is sometimes a cause of regurgitation, apart from valvular disease or dilatation.

¶ IV. Permanent mitral regurgitation produces hypertrophous dilatation of the left cavities of the heart, congestion of the lungs, and lastly dilatation of the right cavities.

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[1] Ludwig and Hesse: quoted by MacAlister: Remarks on the form and mechanism of the heart. Brit. Med. Journ., Oct. 28, 1882.

CHAPTER XXXIII.

MITRAL OBSTRUCTION

¶ I. A MURMUR, after the second sound and before the first, heard louder at or near the apex-beat than over any other part of the heart region, indicates obstruction at the mitral orifice. The murmur is usually præsystolic, that is to say, it is immediately followed, without any interval, by the first sound.[1] The murmur is sometimes diastolic, that is to say, it immediately follows the second sound; there being a distinct interval between the murmur and the first sound.[2] The murmur is sometimes so prolonged as to fill up the whole time between the second and first sounds. The murmur is sometimes double, both diastolic and præsystolic, with a distinct interval between its two parts. The murmur, whether præsystolic or diastolic, is seldom heard at the angle of the scapula. The præsystolic murmur often has a remarkably harsh and whirring character.

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[1] The connection between præsystolic murmurs and mitral obstruction was discovered by Fauvel: Mémoire, etc. Archives gén. de Méd. series iv. vol. i. p. 1. 1843.

[2] It has been plausibly suggested that mitral diastolic murmur is due to the ventricular diastole, and præsystolic murmur to the auricular systole. J. L. Gibson: Lancet for Apr. 19, 1884, p. 730.

¶ II. A præsystolic murmur sometimes passes indistinguishably into a systolic murmur (due to associated regurgitation); sometimes the first sound is well heard between the præsystolic and systolic murmurs; but usually a loud first sound is heard, and no systolic murmur. A diastolic murmur is mostly attended by a systolic murmur. A systolic murmur will sometimes be heard at the angle of the scapula (or indeed all over the back) when the only murmur audible at the apex-beat is præsystolic.[1] The murmur attending mitral obstruction is sometimes undoubtedly systolic only; that is to say, the murmur proper to the obstruction is not heard, but only the concomitant regurgitant murmur. The first sound is loud, and mostly seems to put a sudden stop, as it were, to the præsystolic murmur. The second sound is usually, but not always, inaudible at the apex-beat.

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[1] In a case of this sort, I found that the middle line of the axilla exactly separated the areas of the two murmurs.

¶ III. The murmur of mitral obstruction may be variable: for instance, a præsystolic murmur, followed by a loud first sound and no second sound, may give way for a time to a systolic murmur with first and second sounds; then the præsystolic murmur will reappear, while the systolic murmur and second sound vanish. All murmurs will disappear when the heart is exhausted and unable to expel its contents: in this state, the first sound also usually disappears. The influence of posture upon præsystolic murmurs has been already discussed (p. 147): no examination of the heart is complete if the patient have not been ausculted in both upright and lying posture. Lastly, the murmur sometimes disappears in a manner which cannot be explained.

¶ IV. i. Reduplication of the second sound at the base of the heart (p. 150) is common. ii. The bruit de rappel (p. 150) at the apex is to be deemed a divided diastolic murmur. iii. A thrill often attends the murmur: either præsystolic and running up into the impulse, or diastolic, alternating with the impulse, and accompanied by a back stroke. The thrill is sometimes strong when the murmur is weak. iv. Diastolic aortic murmur is often well heard at the apex, but the diagnosis is seldom difficult when all the signs and symptoms of the case are pondered.

¶ V. A mitral præsystolic murmur signifies permanent structural disease of the valve. The only exception to this rule is the case (first noted by Austin Flint)[1] of præsystolic apex murmur occurring in aortic regurgitation and apart from disease of the mitral valve. The explanation of this fact is uncertain. Mitral and tricuspid obstruction (p. 286) often concur. In mitral obstruction, overfilling of the left auricle and pulmonary congestion are carried to a high degree. Hence hypertrophous dilatation of the left auricle, and dilatation of the right heart.

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[1] In 1862 : See a paper by him in Lancet for March 8, 1884, p. 418. A few other physicians have met with similar cases : see Lees, American Journal Med. Sci., Nov. 1890.

CHAPTER XXXIV.

AORTIC REGURGITATION.

¶ I. A MURMUR, replacing or immediately following the second sound, and heard at the second right interspace and along the sternum, indicates regurgitation through the aortic valve. i. The second sound is sometimes well heard at the second right interspace: even when this is not the case, the second sound is usually heard at the second left interspace. ii. The murmur is often best heard at the lower end of the sternum (and especially a little to the left thereof). iii. The murmur is often, but certainly not always, conducted to the apex: is sometimes heard there better than elsewhere, and thus

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[1] Incompetency of the aortic valves was known to morbid anatomists long before the discovery of auscultation. Hodgkin seems to have been the first to discuss the lesion from the clinical point of view. His papers, "On retroversion of the valves of the aorta," were read before the Hunterian Society on Feb. 21, 1827, and Feb. 18, 1829, and were published in the Lond. Med. Gazette for March 7, 1829. See Wilks: Note, etc.: Guy's Hosp. Reps., series iii. vol. xvi. p. 209. 1871. Hodgkin mentions the diastolic murmur, which Corrigan, in his paper, published in 1832, does not.

simulates the diastolic murmur of mitral obstruction (p. 276). iv. The murmur is often conducted into the arteries near the heart, the carotids and subclavians, so as to be well heard below both clavicles: but this is not always so. v. The murmur is sometimes best heard over the second left interspace or third left cartilage. This means that the heart is displaced to the left; most likely in consequence of elongation of the aorta; the apex-beat will be found to the left of the nipple line. The diagnosis from pulmonary regurgitation depends much upon the characters of the pulse. vi. The murmur sometimes possesses a loud whining or cooing character; and will be audible in all the larger arteries, even so far away as the radials; all over the front of the chest; in the left axilla, and at the angle of the left scapula; by the ear placed nigh to, but not upon, the chest; and lastly, by the patient himself. vii. A systolic basic murmur also is commonly present; or at any rate the first sound is not clear. This systolic murmur is certainly not always due to obstruction at the mouth of the aorta, but is more often the result of mere roughness of the valves, or relative constriction of the orifice, connected with positive dilatation of the ascending aorta. viii. The diastolic murmur may disappear when the heart falls into a state of asystoly, or inability to expel its contents.

¶ II. A diastolic thrill, at the second right interspace, is sometimes present. In the displacement of the heart to the left just spoken of, the thrill may be felt in the second or third left interspace.

¶ III. Hypertrophy of the left ventricle usually ensues: hence the heaving impulse, mostly but not always present: hence the frequency of dilatation of the ascending aorta; the elongation of the arteries; and the intensity of the arterial systolic murmur. Diastolic arterial murmur (p. 162) is often present. But of all arterial signs, a jerking pulse[1] is the most important. It may be present even when there is no hypertrophy of the ventricle. Nay more, the heaving impulse, and all the arterial signs are sometimes absent, when a distinct diastolic murmur in the aortic region would

[1] "Après avoir remarqué l'abattement de ses yeux, la bouffissure, et la pâleur de son visage, j'examinai son pouls qui me parut fort plein, fort vîte, dur, inégal, et si fort que l'artère de l'un et l'autre bras frappait le bout de mes doigts autant que l'auroit fait une corde fort tendue et violemment ébranlée." Histoire de Jean Chifort (case of disease of aortic sigmoids): Vieussens. Œuvres françoises, 1715.

seem to render the nature of the disease quite certain. Yet, in cases of this kind, it may happen that there is no regurgitation, no disease of the sigmoids, and no dilatation of the ring to which they are attached: nothing more than dilatation and rigidity of the ascending aorta.[1] Pulsatile flushing of the lips, finger-nails and other parts (capillary pulse) sometimes accompanies the jerking pulse.

¶ IV. i. A result of aortic regurgitation, less common than hypertrophy of the left ventricle, is dilatation thereof. In which case the mitral orifice partakes in the dilatation; and becomes regurgitant; the cusps remaining unchanged: all the results of mitral regurgitation follow. The aortic and mitral valves are often simultaneously diseased, so that the flaps of both valves are altered in structure. ii. The occurrence of præsystolic apex murmur in cases of aortic regurgitation, and apart from disease of the mitral valve, has already been mentioned (p. 279). iii. When mitral obstruction and aortic regurgitation concur, the diagnosis of both lesions is often difficult; the signs of one tend to override those of the other.

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[1] See Finlayson: Brit. Med. Journal, Feb. 28, 1885, p. 426.

CHAPTER XXXV.

AORTIC OBSTRUCTION.

¶ I. The murmur present in cases of aortic obstruction is systolic, and heard loudest in the second right interspace. But many of the murmurs commonly called functional or inorganic possess these characters (p. 292). Wherefore it is necessary to the diagnosis of aortic obstruction that the murmur be loud and long, and attended by a heaving impulse indicative of hypertrophy of the left ventricle,[1] and by a pulse which is small even when the heart is beating strongly: moreover the signs of aneurysm must be absent.

¶ II. A systolic thrill, felt on the right side of the base of the heart, is often present.

¶ III. Aortic obstruction is usually complicated with regurgitation; even when there is no diastolic murmur, the aortic second sound is muffled, weakened, or absent.

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[1] Traube (Gesammelte Beiträge: vol. ii. p. 831) has published two cases of uncomplicated aortic obstruction in which the heart's impulse was imperceptible: and this weakened impulse (provided that no other cause thereof be present) he considers to be important evidence of aortic obstruction.

CHAPTER XXXVI.

TRICUSPID REGURGITATION.

¶ I. A MURMUR, systolic, and heard best over the lower part of the sternum, or a little to the left thereof, may be due to tricuspid regurgitation. The valvular disease is mostly consequent upon dilatation of the right heart: and a dilated heart usually contracts weakly: hence, in many cases of tricuspid regurgitation, there is no corresponding murmur. The murmur is not conducted far from the spot mentioned; is not heard in the armpit or at the angle of the left scapula: and sometimes is not heard in the upright, but in the lying posture only.

¶ II. More important are the signs which the jugular veins (p. 166), and the liver (p. 172) afford. Yet it must be confessed that jugular pulsation may be quite invisible, even in great incompetency of the tricuspid valves with hypertrophy of the right ventricle: the valve at the mouth of the vein being probably competent in this case. A systolic thrill at the epigastrium is said to have been present in a few cases. The associated dilatation of the right heart cannot always be made out by percussion, because of pulmonary emphysema.

CHAPTER XXXVII.

TRICUSPID OBSTRUCTION.

¶ I. A PRÆSYSTOLIC murmur, heard over a space defined by a line drawn from the fourth right chondrosternal joint downwards along the right margin of sternum to the tip of the ensiform cartilage, thence to the sixth left chondrosternal joint and thence to the fourth right chondrosternal joint, is the sign of tricuspid obstruction.[1]

¶ II. Præsystolic thrill may be felt over the same area. The heart's percussion dulness is much extended to the right, because of the dilatation of the right auricle consequent upon the tricuspid disease. The jugular veins are distended, but seldom pulsate.

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[1] Bedford Fenwick: Lancet, 1881, vol. i. pp. 653 sqq. Tricuspid obstruction is always attended by mitral obstruction: and, in half the cases, by aortic disease, obstructive or regurgitant. Eight-ninths of the cases of tricuspid obstruction are female.

CHAPTER XXXVIII.

PULMONARY REGURGITATION.

¶ I. THIS, the most uncommon of all valvular diseases,[1] is attended by a diastolic murmur, heard loudest in the second left interspace and along the sternum, or loudest sometimes at the ensiform cartilage. Simplicity of doctrine requires that this should be the area of the murmur, but experience tells that the second right interspace may possibly be the place of greatest loudness.[2] Moreover, aortic regurgitant murmurs are sometimes loudest over the second left interspace (p. 281). Wherefore diagnosis between pulmonary and aortic reflux is not so easy as might at first appear. The two regurgitations may even be associated. But when either semilunar valve alone regurgitates, the presence or absence of the jerking pulse and of the other arterial signs of aortic disease, is the means of attaining a correct opinion.

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[1] Barié: Arch. gén. de méd. for 1891, vols. i. and ii.

[2] Duckworth: Clinical Trans., vol. xxi. p. 18.

¶ II. A diastolic thrill is sometimes present. A systolic murmur, not always due to obstruction, may occur in pulmonary regurgitation just as in the corresponding aortic disease.

¶ III. Dilatation of the right ventricle, and impeded systemic venous circulation, with dilatation and pulsation of the jugulars, are possible consequences. Dilatation of the pulmonary artery is sometimes indicated by pulsation of the second and third left intercostal spaces.

CHAPTER XXXIX.

PULMONARY OBSTRUCTION.

THE murmur of pulmonary obstruction is systolic, and heard loudest on the third rib, or in the second or third left interspace, close to the sternum.[1] A systolic thrill may be felt at the same spot. A loud murmur will be conducted far in all directions, even to the back. Hypertrophy of the right ventricle supervenes; so that it is not until the obstruction becomes great, or the heart's contractions begin to fail in vigour, that there is any stagnation in the general venous system.

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[1] Ormerod: Edin. Med. and Surg. Journ. no. 166. 1846.

CHAPTER XL.

MURMURS OF UNCERTAIN NATURE.

FROM the earliest times of auscultation it has been known that some murmurs are temporary in duration and not indicative of permanent disease of the heart.[1] These murmurs are always systolic, and taken by themselves alone, they cannot be distinguished from systolic murmurs due to valvular disease; for the areas over which temporary murmurs are heard loudest, are the same as those which have been allotted to murmurs indicative of permanent valvular disease.

Temporary murmur is apt to accompany—i. Blood-dyscrasiæ: especially anæmia, however produced; sometimes jaundice. ii. Palpitations of the heart. iii. Pressure upon the chest over the base of the heart (p. 147). iv. Pregnancy. v. A loud murmur sometimes springs up in dying persons, the heart being found natural after death.

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[1] These murmurs have been called functional, anæmic, hæmic murmurs; but no name both apt and comprehensive has yet been devised for them, or perhaps can be.

I. Pulmonary and aortic murmurs. The common temporary murmur has the characters of obstruction at a semilunar orifice, especially the pulmonary.[1] These murmurs are heard, sometimes over a very limited and sometimes over a very extensive space : sometimes in the back, especially between the left scapula and the spine :[2] they are louder in the recumbent than in the erect posture, and may even be audible in the recumbent posture alone. Strong beating of the heart will sometimes make them louder, or will even bring out a murmur which is inaudible when the heart is quiet. Pulsation in the second interspace is sometimes felt, and, in rare cases, is so strong as to raise a suspicion of aneurysm. Signs of dilatation of the heart (apex-beat displaced to left and increased extent of percussion dulness) are usually present.

II. Mitral and tricuspid murmurs are likewise attended by signs of dilated heart : tricuspid murmurs by distension and pulsation of the jugular veins also. Anæmia is a common concomitant of these murmurs: jaundice may be attended by a mitral systolic murmur,[1] and pregnancy by a tricuspid systolic murmur.[2] Debility of the heart, with secondary dilatation of its chambers and apertures, with relaxation of its valvular sphincters, and consequent valvular regurgitation, seems to afford a satisfactory explanation of most temporary murmurs of this kind.

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[1] Foxwell : Causation of functional cardiac murmurs, 1899 : attributes the pulmonary murmur to dilatation of the right conus arteriosus and of the pulmonary artery.

[2] A. E. Garrod : St. Barthol. Hosp. Reports : vol. xxvii. p. 34.

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[1] Legg: On the bile, etc., 1880: p. 306.

[2] Money: Med.-Chir. Trans., vol. lxv. p. 87.

CHAPTER XLI.

MALFORMATIONS OF THE HEART.

CONGENITAL malformation of the heart is usually discovered easily enough : but not so the precise kind of malformation. We may attempt a diagnosis for which our data are mostly insufficient, but we shall seldom advance beyond a guess.

i. The heart region is sometimes bulged, especially in older patients. ii. The impulse is often heaving, often diffused, sometimes hardly palpable. iii. The apex-beat is commonly displaced to the left, and is also lower than natural. iv. Extension of percussion dulness to the right is not uncommon. v. A murmur, systolic, and heard loudest over the pulmonary region (from the second to the third left interspace, close to the sternum), is common : the murmur is very local, or heard over the heart region, or conducted more or less extensively over the chest. Much less common murmurs are these : diastolic in pulmonary region (p. 152), with or without systolic: systolic at left apex: systolic at epigastrium, or just above and to the left thereof: and probably others which I have not met with. Sometimes the murmur disappears just before death. Sometimes there is no murmur at all. vi. A thrill often, not always, coincides with the murmur; being best felt where and when the murmur is loudest; and being very local or conducted more or less widely. vii. Signs in the jugular veins are uncommon.

CHAPTER XLII.

MEDIASTINAL TUMOURS:

INCLUDING mediastinal abscesses, and enlargement of the thymus, or of the bronchial glands. i. A large and quickly-growing tumour, or abscess, sometimes causes protuberance of the front of the chest near the sternum: and this swelling may pulsate so as to simulate aneurysm. ii. The heart may be displaced downwards, upwards, or to either side; or may be pressed forwards. iii. Vocal vibration diminished or absent over the area of dulness to percussion. iv. Great percussion dulness and resistence will be found over a tumour which is in contact with the chest-wall, either in front (behind, and alongside the sternum) or behind, displacing or involving one or other lung: except that small tumours, connected with the trachea or a bronchus, will afford a clear, tracheal note. v. The tumour, when small, may or may not conduct sounds generated in its vicinity; bronchial breathing, bronchophony, and the heart's sounds: when large, no sound will be heard over the tumour. vi. Sometimes a systolic murmur at the base of the heart is produced by pressure, and so an aneurysm may be simulated. vii. Signs of obstruction to the large veins are common. viii. Signs of obstruction to the trachea or a bronchus; of collapse of the lung; of destructive or gangrenous pneumonia; of pleurisy with effusion; and of pericarditis; often complicate the signs proper to tumour. ix. Tumour, or abscess, or secondarily enlarged glands, may be felt above the collar bone. x. There are no physical signs of tuberculous disease of the bronchial glands (strictly so-called), namely, those in the roots of the lungs and the bifurcation of the trachea: whereas disease of the tracheal glands often affords dulness to percussion alongside the manubrium sterni, and sometimes a swelling to be felt deep in the neck, behind the clavicle.[1]

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[1] On Emphysema of the Mediastinum: see Müller, Berlin klin. Wochenschr. Mar. 12, 1888: p. 205.

CHAPTER XLIII.

ANEURYSM OF THE THORACIC AORTA.

§ I. Aortic aneurysm must be large enough to come into contact with the chest-walls in order to yield any physical signs directly dependent upon itself. Earliest to appear are the signs afforded by palpation and percussion.[1]

¶ I. Palpation detects pulsations and thrills. The Pulsation is systolic, synchronous with the latter part of the heart's impulse. A slight diastolic shock, or even a strong diastolic impulse, can sometimes be felt. When the aneurysm contains much coagulum, the pulsation is weak or even absent. i. Aneurysm of the ascending aorta usually springs from the right or convex side of the vessel, and in this case, touches the chest-wall first in the second right interspace close to the sternum : as the tumour enlarges, the pulsation extends upwards towards the clavicle and manubrium sterni, or downwards along the right margin of the sternum, haply as low as the fourth, or even fifth, interspace. Aneurysm, which springs from the left or concave side of the ascending aorta, produces a tumour to the left of the sternum, between it and the left nipple line, and between the second and fifth left costal cartilages, especially about the third cartilage. The aneurysm tends to thrust the heart over into the right side of the chest horizontally ; and so much so, when the aneurysm is very big, that it comes to occupy the natural position of the heart, while the heart lies altogether to the right of the sternum.[1] ii. Aneurysm of the transverse aorta first comes to the surface behind the upper part of the sternum, and afterwards extends far away towards the left. iii. Aneurysm of the descending aorta, at its upper part, points below the first left rib, and thence extends downwards to the second space. iv. Aneurysm of the lower part of the thoracic aorta lies upon the left side of the dorsal vertebræ and may cause pulsation there. The pulsation is sometimes attended by systolic or diastolic Thrill.

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[1] Aneurysm of a Valsalvian sinus seldom affords distinctive physical signs. Pulsating tumour is present in about seven per cent. of the cases, one half being to the right, and one half to the left of the sternum. Murmur is common, and due to regurgitation through the aortic or pulmonary valves. Sibson: Medical Anatomy, fasciculus v. 1858 : p. 2.

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[1] St. Bart. Hosp. Reps. vol. xxx. p. 1.

¶ II. Percussion discovers dulness in the same situations: namely, behind the second and third ribs along the right side of the sternum; behind the manubrium sterni; to the left side of the sternum; and along the left side of the dorsal vertebræ. The aneurysmal dulness is sometimes continuous with the cardiac dulness, sometimes not.

¶ III. Inspection sometimes discovers a tumour in a position corresponding with that of the palpation and percussion signs. In shape the tumour is hemispherical, except that it is sometimes uneven when constricted by resistent fibres in overlying parts, or when the aneurysm itself is nodular. Aneurysm of the descending aorta may be so great as to push the scapula outwards.

¶ IV. Auscultation does not afford much help to diagnosis: there will be a first and second sound, or a systolic and diastolic murmur, or a first sound and diastolic murmur, or a systolic murmur and a second sound, or a single sound or murmur, or no kind of sound at all. The sounds are probably those of the heart conducted: the systolic murmur is due to the passage of blood through a relatively narrow mouth into a wider cavity, whether an aneurysmal sac or a dilated aorta ; the diastolic murmur is either a conducted murmur produced by regurgitation at the mouth of the aorta, or is due to the passage of blood out of a sac during its contraction, or is inexplicable. Systolic murmur may be heard over the trachea ; is sometimes louder over the trachea than at the base of the heart ; and may, in rare cases, be heard over the trachea and not over any part of the chest.[1] Murmurs, when loud, may be conducted far away, along the arteries.

§ II. Uniform dilatation of the ascending aorta is attended by extension of percussion dulness to the right of the sternum (level with the second and third cartilages) and behind the manubrium sterni. Elongation of the aortic arch forces the base of the heart downwards and to the left ; so that the apex-beat comes to be lower and more external than natural.[2]

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[1] Drummond : Brit. Med. Journ. Oct. 21, 1882 : p. 773.

[2] W. S. Oliver was the first to describe a sign which has since been called Tracheal Tugging. He tells us to "place the patient in the erect position, and direct him to close his mouth and elevate his chin to the fullest extent, then grasp the cricoid cartilage between the

§ III. Aneurysmal tumour, like other mediastinal tumours, will compress neighbouring viscera. i. Pressure upon the trachea causes noisy breathing (stridor), audible at a distance from the patient. ii. Pressure upon the bronchus, commonly the right, also causes stridor: by auscultation, the sound will be loudest where the second rib joins the sternum.[1] iii. Pressure upon the root of the lung produces collapse, dilated bronchi, bronchitis, or destructive pneumonia. iv. Pressure upon the spongy structure of the lung produces relaxation or collapse of the part involved. v. Pressure upon the vena cava superior or vena innominata is attended by the signs of venous obstruction. vi. The heart is liable to sundry displacements, thus: dilatation of the convexity of the ascending aorta usually implies elongation thereof, whereby the heart is

finger and thumb, and use gentle upward pressure on it, when, if dilatation or aneurysm exist, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." (Lancet, Sep. 21, 1878: p. 406.) A slight degree of tugging cannot be depended upon as a sign of aneurysm: but well-marked tugging is believed by R. L. Macdonnell (Lancet, Mar. 1891: pp. 535, 650) to indicate aneurysm of the transverse aorta, so situated as to press downwards upon the left bronchus, or adjacent part of the trachea.

[1] See note on p. 157, concerning pulse-breath.

forced downwards and to the left : a large tumour of the transverse aorta produces the same effect : aneurysm of the concavity of the ascending aorta thrusts the heart to the right : a very large tumour of the descending aorta will push the heart towards the right : a tumour behind the heart will press it against the front wall of the chest.

§ IV. Anastomotic aortic aneurysm ; that is to say, which opens into a neighbouring part of the circulating system. i. Into the vena cava superior : the signs are indicative of venous obstruction and of aneurysm of the ascending aorta ; in some cases there have been observed a systolic thrill and murmur in the veins of the neck, and a thrill and loud systolic murmur (or a continuous hum, but loudest during the systole) at or below the second right cartilage. ii. Into the right auricle ; no signs more definite than those of an aortic aneurysm and of venous obstruction. iii. Into the right ventricle ; no definite signs. iv. Into the pulmonary artery : systolic and long diastolic murmur at fourth left rib ; no natural sounds there ; diastolic thrill at the same spot : diastolic murmur inaudible at the apex-beat ; pulsation and thrill in the carotids (p. 152).

§ V. Aortic aneurysms are simulated by pulsating mediastinal tumours or abscesses (p. 295), and pulsating empyema (p. 231). Aneurysms, which do not pulsate and which are silent, simulate tumours of another kind.

CHAPTER XLIV.

OTHER INTRATHORACIC ANEURYSMS.

§ I.—ANEURYSM OF THE INNOMINATE ARTERY.

¶ I. DILATATION of that part of the artery which is nearest to the heart is always associated with aneurysm of the ascending aorta, and is not attended by any distinctive signs.

¶ II. Dilatation of the distal part of the artery affords these signs following: i. A pulsating tumour, which comes up, between the origins of the sterno-mastoid muscle, from behind the right sterno-clavicular articulation, is due to an innominate aneurysm: provided there be no signs of aortic disease; for aortic aneurysm may rise into the same position. The head of the clavicle may be dislocated. ii. If there be, over the tumour, a systolic murmur, it is conducted into the right carotid, but not into the left. iii. Pressure upon one or both of the innominate veins is common.[1]

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[1] Cockle: Medico-Chir. Trans., vol. l. p. 459. 1867.

§ II.—ANEURYSM OF THE PULMONARY ARTERY.

A very uncommon disease, seldom discoverable during life. These signs are said to have been noted: i. More or less swelling about the second and third left interspaces, nigh the sternum. ii. Systolic, and sometimes diastolic impulse there. iii. Some dulness to percussion. iv. Very loud sounds or murmurs, especially systolic, in the same place. But all these signs are sometimes afforded by aneurysm of the ascending aorta (p. 298). Consecutive lesions relate to the right, rather than to the left, heart.

INDEX.

Abscess : mediastinal. *See* Mediastinal. Subdiaphragmatic and hepatic, 211, 226, 228 : diagnosis from pleural effusion, 182 : from pneumothorax, 211

Accentuation of pulmonary second sound, 136

Actinomycosis of lung, 254 : diagnosis from pleural effusion, 225

Adherent pericardium. *See* Pericardial adhesion

Adherent pleura. *See* Pleural adhesion

Aegophony, 100, 107

Aërifaction of lung, 105, 109, 120

Alar chest, 11

Amphoric auscultation sounds, 128 : hum, 128, 131 : percussion tones, 65, 70, 85 : vocal resonance, 106. *See* Bell sound, Metallic tinkle, Splashing sound

Anæmia. *See* Cachexia

Anapnograph, 35

Anastomotic aortic aneurysm. *See* Aneurysm

ANDRY, cyrtometer, 8

Aneurysm : bulging of chest, 31 : murmurs, 160 : compression of bronchi, 239 : simulated by other mediastinal tumours, 295 : by cancer of lung, 251 : by pulsating empyema, 230, 231 : anastomotic of aorta, 53, 152, 302 : aortic, 297 : of innominate artery, 304 : intrapulmonary, 178, 247 : of pulmonary artery, 305 : of Valsalvian sinus, 297

Angular curvature. *See* Kyphosis

Angulus Ludovici, 10
Aorta. *See* Aneurysm, Dilatation, Elongation
Aortic murmurs : conduction of, 142, 143, 160, 163, 165, 280
Aortic obstruction, 284 : thrill, 52
Aortic orifice, situation, 142
Aortic regurgitation, 280 : thrill, 52 : arterial murmur, 160
ARETAEUS : alar chest, 12
Arteries, auscultation and murmurs, 160
Articulation of voice, 102
Ascites : effect on respiratory movements, 38 : displaces heart, 41
Asthma, 196 : expiratory dyspnœa, 37
Asystoly, 148, 274, 278, 282
Atrophy of lungs, 195
AUENBRUGGER discovers percussion of chest, 55: method of percussion, 58 : pulmonary percussion tone, 63: nomenclature, 64 : dictum, 81 : epigastric swelling, 175 : pericardial effusion, 268
Auricle, impulse, 42 : rupture of aneurysm into, 302
Auscultation, 88 : history, 88: methods, 91 : lungs, 95 : voice, 96 : breathing, 111 : pleura, 125 : heart, 133 : pericardium, 152 : arteries, 160: jugular veins, 170 : œsophagus, 183: aneurysms, 299. *See* Table of Contents

BAAS, tuning fork in percussion, 60
Back stroke of heart, 50
BACON, quoted, 91
BARIE, pulmonary regurgitation, 287
BEAU, theory of breathing sounds, 116: expiratory murmur in femoral vein, 171
Bell sound, 129, 131
BENNETT, intrathoracic cancer, 250

Bergeon, murmurs, 95
Bird, hydatids of lung, 181, 252
Bouillaud, bruit de rappel, 150 : diastolic femoral murmur, 162
Bricheteau, water-wheel sound, 155
Bronchi, compression, 239, 301 : obstructed, shape of chest, 31. *See* Dilatation, Plugging
Bronchial breathing, 112, 117, 118, 119
Bronchial glands, enlargement, 296
Bronchitis, plastic, 187. *See* Catarrh
Bronchophony, 97, 104, 109
Brown induration of lungs, 191
Bruit de rappel, 150
Bruit skodique, 63
Bulging of chest, 31 : in hydatids, 252 : in aneurysm, 298, 299, 304, 305 : in pleural effusion, 218, 228, 230, 231

Cachexia : arterial murmurs, 161 : venous murmurs, 170 : murmur at base of heart, 151, 291 : mitral murmur, 292 : tricuspid murmur, 292. *See* Murmurs of doubtful nature
Caelius Aurelianus, rales, 88 : bulging of chest in bronchitis, 186
Cancer of lung, 249 : shape of chest, 31 : movements of chest, 38 : vocal thrill, 47 : diagnosis from pleural effusion, 224
Cantering action of heart, 151
Cardiac. *See* Heart
Cardiograph, 49
Carnification of lung, 200, 257
Carswell, second sound of heart, 134
Catarrh of lungs, 184 : shape of chest, 15, 16, 186 : movements of chest, 37 : in acute tuberculosis, 242 : simulated by pleurisy, 205

Cavernous breathing, 114, 117: rales, 124: signs in pleurisy, 205

Cavity in lung, signs of, 245: percussion sound, 83: bronchophony, 106, 109: bronchial breathing, 120: amphoric signs, 128: pulsatile sounds, 157: vascular murmurs, 178

CHAUVEAU, cardiograph, 49: venous hum, 170

Chest, shape, 4: movements, 34: puncture, 179. *See* Table of Contents

Children, shape of chest, 6: position of heart, 39: percussion note, 66, 73, 77

Chlorosis. *See* Cachexia

Cirrhosis of lung, 256, 200: shape of chest, 30: displaces heart, 42: diagnosis from pleural effusion, 225: in phthisis, 245: in dilated bronchi, 255: following collapse, 200

CLAR, life of Auenbrugger, 56

Clearness of percussion tones, 64, 82: of vocal resonance, 97

COCKLE, aneurysm of innominate artery, 304

Collapse of lung, 198: shape of chest, 31: crepitant rale, 119, 122: in pulmonary catarrh, 186: in bronchial obstruction, 203: in dilated heart, 262: in pericardial effusion, 268: in aneurysm, 301: diagnosis from pleural effusion, 225

COLLIN, pericardial friction, 138

COMBY, pulsating empyema, 229

Complementary space of pleura, 174

Conduction of sound by lung, 104: of murmurs, 142: of pulmonary and pleural sounds, 182

Congestion of lungs, 191

Consonance, 69

Constrictive murmurs, 140

Conus arteriosus, impulse, 42

Convection of murmurs, 143

COPLAND, cup-like hollow, 173

CORVISART, discovers valvular thrill, 51 : discovers Auenbrugger, 56 : method of percussion, 59 : percussion resistence, 74

Cough, thrill, 47 : resonance, 110

COUPLAND, subphrenic abscess, 212

Cracked-pot sound, 65, 72 : in pleural effusion, 216 : in pneumonia, 235 : in phthisis, 247 : in cancer, 250 : in pneumopericardium, 271

Creaking rales, 124 : in phthisis, 247

Crepitant rale, 119, 121 : pulsatile, 158 : in œdema of lung, 189 ; in collapse of lung, 198 : in pneumonia, 234 : in phthisis, 247. *See* Dry crepitant rale

Croup, cup-like depression, 32

Cry, resonance, 110 : in pneumonia, 236

Cup-shaped depression, 32 : in pleurisy, 205, 223

Cyrtometer, 8

DAMOISEAU, pleural effusion, 222

DAVIES, Thos., puncture of chest, 179

Deep percussion, 75

Deformities of chest, 10

Destructive pneumonia, 203, 239 : diagnosis from pleurisy, 225

Diaphragm, position, 173 : in health, 173 : in disease, 174 : depression, 175 : elevation, 175 : rupture, 210 : relation to transverse constriction of chest, 16 : paralysis, 35 : congenital deficiency, 211. *See* Abscess subdiaphragmatic

Diastolic, definition, 38 : thrills, 52, 53, 278, 282, 288 : impulse, 50, 297 : heart murmurs, 144, 146, 276, 280, 289 : arterial murmurs, 152, 162 : venous murmur, 171

Dilatation of aorta, 152, 282, 300

Dilatation of bronchi, 255 : in destructive pneumonia, 239

Dilatation of heart, 260 : shape of chest, 31 : epigastric pulsation, 172 : diagnosis from pericardial effusion, 268. *See* Enlargement of heart
Diphtheritic bronchitis, 187, 238
Displacement of heart. *See* Heart
DOUBLE, respiration bruissante, 88
DOUGLAS, heart murmur, 89
DRUMMOND, pulse-breath, 157, 300
Dry crepitant rale with great bubbles, 125
DUCHENNE, shoulder-blade friction, 133
DUCKWORTH, pulmonary regurgitation, 287
Ductus arteriosus, murmur in, 152
Dulness of percussion sounds, 72, 83
Duration of percussion tones, 62
DUROZIEZ, quoted, 134 : diastolic arterial murmur, 162
Dyspnœa, inspiratory, 36, 190 : expiratory, 37 : in asthma, 196

ELLIOTSON, heart murmurs, 147, 273
Elongation of aorta, 259, 281, 300
Emphysema of lungs, 193 : shape of chest, 22 : movements of chest, 37 : friction, 126, 127 : acute, 186 : associated with phthisis, 247
Empyema, 229 : shape of chest, 28, 31 : movement of chest, 37 : fistulous, 209 : loculated, 227 : pulsating, 229 : diagnosis from destructive pneumonia, 239 : after pneumonia, 237
Enlargement of heart : position of impulse, 41, 42 : depression of diaphragm, 175. *See* Dilatation, Hypertrophy
Epigastric pulsation, 171 : recession, 173
Expiration, shape of chest, 5
Expiratory dyspnœa. *See* Dyspnœa
Exploratory puncture of chest, 179

Fauvel, præsystolic murmurs, 276
Femoral vein, expiratory murmur in, 171
Fenwick, tricuspid obstruction, 286
Finlayson, rigid aorta, 283
Fistulous empyema, 209
Flames, sensitive, 60
Flat chest, 12
Flint, præsystolic apex murmur, 279
Fluctuation, pericardial, 54, 267 : pleural, 47
Fluid veins, 94
Foramen ovale, murmurs at, 141
Forbes, translation of Auenbrugger, 56
Forestus, heart sounds, 89
Fournet, froissement pulmonaire, 125
Friction, pericardial, 53, 138, 263 : simulated by pleural friction, 127, 264 : pleural, 47, 125 : pulsatile, 158 : simulated by peritoneal friction, 132 : shoulder-blade, 133 : shoulder-joint, 132
Friedreich, venous murmurs, 171 : expiratory murmur in femoral vein, 171 : pulsation of liver, 172
Fulness of percussion sounds, 62

Gairdner, friction of emphysema, 127 : tricuspid obstruction, 53
Galen, alar chest, 12 : pericardial fluctuation, 54
Gangrene of lung, 240. *See* Destructive pneumonia
Garrod, A. E. anæmic murmurs, 291
Gerhardt, resonators, 60 : intrapulmonary aneurysmal murmur, 178
Gibson, murmurs of mitral obstruction, 276
Gordon, posture and heart murmurs, 147
Gowers, shoulder-joint friction, 132
Graves, depressed sternum, 33 : pulsating pneumonia, 237 : pericardial effusion, 268 : pneumopericardium, 271

GREENFIELD, hydatid of lung, 253
Grey induration of lung. *See* Cirrhosis
Gurgling rale, 123

Hæmorrhage from and into lung, 192
Hæmorrhagic infarctus of lung, 192
Hæmothorax. *See* Pleural effusion
HALDANE, tricuspid obstruction, 53
HALL, De Havilland, intrathoracic sarcoma, 224
HALL, Radclyffe, pulse-breath, 157
HAMBURGER, auscultation of œsophagus, 183
HARRISON, sulcus, 16 : pectoriloquy, 100
HARVEY, heart sounds, 89
Heart, auscultation, 133 : bulging, 31 : displacement, 41, 42 : palpitation, 49 : percussion, 79, 86 : sounds, 135 : thrills, 51. *See* Aortic obstruction and regurgitation, Asystoly, Dilatation, Enlargement, Hypertrophy, Impulse, Malformations, Mitral obstruction and regurgitation, Movements, Murmurs, Pulmonary obstruction and regurgitation, Reduplication, Second sound, Tricuspid obstruction and regurgitation, Valvular
Hernia of lung, 31
HIPPOCRATES, alar chest, 12 : auscultation by, 88
History of aortic regurgitation, 280 : auscultation, 88 : friction sound, 125, 138 : mitral obstruction, 276 : percussion, 55 : physical signs of heart, 133, 138 : tricuspid regurgitation, 167
HOBBES, on signs, 1
HODGKIN, aortic regurgitant murmur, 280
HOFFMANN, auscultation of œsophagus, 183
HONORÉ, pleural friction, 126
Hooping-cough, pigeon breast, 17 : cupping, 32
HOPE, back stroke, 50 : regurgitant murmurs, 273
Humoral percussion-note, 74

Hydatid thrill, 75
Hydatids of lung, 252 : shape of chest, 28, 31 : diagnosis from pleural effusion, 224 : puncture, 181
Hydropericardium. *See* Pericardial effusion
Hydropneumopericardium, 155, 271
Hydropneumothorax, 48, 206, 253
Hydrothorax, 213 : with œdema of lungs, 190
Hypertrophy of heart, 258, 282 : bulging of chest, 31 : recession of chest, 43 : impulse, 49, 50 : arterial murmur, 161. *See* Dilatation, Enlargement

IMMERMANN, intrapulmonary vascular murmurs, 178
Impulse of aneurysms : of aorta, 297 : innominata, 304 : pulmonary artery, 305
Impulse of heart : systolic of apex, 39, 41 : of conus arteriosus, 42 : of right auricle, 43 : diastolic, 50 : præsystolic, 50
Induration of lung, brown, 191 : grey, 256. *See* Cirrhosis
Infarctus of lung, hæmorrhagic, 192 : pyæmic, 241
Innominate artery, aneurysm. *See* Aneurysm
Inspection of chest, 4. *See* Table of Contents
Inspection of jugular veins, 163
Inspiration, shape of chest in, 5 : non-expansive, 37
Inspiratory dyspnœa. *See* Dyspnœa
Intrapulmonary aneurysm. *See* Aneurysm

Jerking breathing, 112
JOHNSON, James, regurgitant murmurs, 273
Jugular veins, inspection, 163 : pulsation, 164 : auscultation, 170

KOLISKO, metallic tinkle, 131
Kyphosis, shape of chest, 24

LAENNEC, M. vesicular breathing, 111

LAENNEC, R. T. H. discovers auscultation, 90 : ægophony, 100, 107 : amphoric hum, 128 : bell sound, 129 : bronchial breathing, 112 : bronchophony, 104 : carnification of lung, 200 : cavernous breathing, 114 : cavernous rale, 124 : cracked-pot sound, 66 : dry crepitant rale with big bubbles, 125 : heart's impulse, 49 : heart murmurs, 133 : metallic jingle, 158 : metallic tinkle, 129, 131 : obscure rale, 123 : pectoriloquy, 99 : pleural friction, 126, 127 : puerile breathing, 112 : puff, 114 : pulsatile pulmonary sounds, 156 : pulsating pneumonia, 236 : rales, 121 : rupture of diaphragm, 211 : tubular cough, 114 : valvular thrill, 51 : veiled puff, 115

LANCISI, pulsating jugular veins, 167

Laryngeal obstruction, depression of sternum, 32 : simulated by hydrothorax, 213

Laryngeal sounds of breathing, 116

LATHAM, quoted, 113 : auscultation of heart, 134, 138

LEES, quoted, 279

LEGG, murmur in jaundice, 292

Liver, depression, 175 : percussion dulness, 78 : pulsation, 174 : relation to constriction of thorax, 16 : tumours, diagnosis from pleural effusion, 225. *See* Abscess, Subdiaphragmatic

Lobular pneumonia. *See* Pneumonia

Loculated empyema. *See* Empyema.

Loculated pneumothorax. *See* Pneumothorax

Loudness of sound, 61

LOUIS, angulus Ludovici, 10 : on emphysematous crepitation, 125

LUDWIG and HESSE, closure of cuspid orifices, 275

Lungs, auscultation, 95 : conducting power, 104 : elasticity, 179 : palpation, 45 : percussion, 71, 77, 82 : relaxed, 71, 82 : vascular murmurs in, 177. *See*

Actinomycosis, Aërifaction, Aneurysms, Atrophy, Brown induration, Catarrh, Cavity, Cirrhosis, Collapse, Congestion, Diphtheritic bronchitis, Emphysema, Gangrene, Grey induration, Hæmorrhage, Hernia, Hydatids, Induration, Infarctus, Œdema, Phthisis, Plugging, Pneumoconiosis, Pneumonia, Solidification, Syphilis, Tuberculosis

MACDONNELL, tracheal tugging, 300
MACKENZIE, auscultation of œsophagus, 183
MAHOT, hepatic pulsation, 172
Malformation of heart, 293
MAREY, cardiograph, 49
MARKHAM, dilated auricle, 43
MAYOR, sounds of fœtal heart, 89
Measurement of chest, 9, 27. *See* Cyrtometer
Mediastinal abscess, 295: tumour, 295 : emphysema, 296
Mediastinum, position, 175
Membranous bronchitis, 187
MERCIER, hydatid of lung, 253
Metallic jingle, 158
Metallic ring, 65, 70, 72, 85, 129
Metallic tinkle, 129, 131
Mitral murmurs, 143 : valve, position, 142
Mitral obstruction, 276 : thrill, 53: and aortic regurgitation, 283 : and tricuspid obstruction, 286
Mitral regurgitation, 143, 273: thrill, 52 : with aortic regurgitation, 283
MONEY, heart murmurs in pregnancy, 292
MONTAIGNE, quoted, 90
MOORE, hypertrophy of heart, 31
MORTON, shapes of chest, 14
Movements of respiration, 34 : of heart, 38, 48
Mucous rale, 123
Muffled percussion sounds, 64, 83

MÜLLER, emphysema of mediastinum, 296
Murmurs, in general, 94 : aneurysmal, 160, 299 : arterial, 160 : cardiac, 139 : subclavian, 161 : vascular in lungs, 177 : venous, 170
Murmurs of doubtful nature; at base of heart, 151, 223 : in general, 290
Muscular rumble, 133

Nasal bronchophony, 100, 108

Obscure rale, 123
Obstructive murmurs, 140
Œdema of lungs, 189, 213 : crepitant rale, 121
Œsophagus, auscultation, 183
Old people, shape of chest, 8 : position of heart, 40
Oligaemia. *See* Cachexia
OLIVER, tracheal tugging, 300
ORMEROD, E. L. pulmonary obstructive murmur, 289
Osteal percussion tone, 63

Palpation, 45. *See* Table of Contents
Parasternal line, 10
PARROT, venous murmurs, 171
Pectoriloquy, 99, 104
Percussion, 55. *See* Table of Contents
Pericardial adhesion, 272 : recession of chest wall, 43 : recession of epigastrium, 173 : cup-shaped depression, 32
Pericardial effusion, 265 : bulging of chest, 31 : movements of chest, 38 : fluctuation, 54 : puncture, 180
Pericardial friction. *See* Friction
Pericarditis, 263 : in empyema, 228
Peritonitis, movements of chest, 38 : friction, 132
PHILLIPS, murmur at base of heart in pleural effusion, 224

Phthinoid chests, 12, 13

Phthisis pulmonalis, 244 : shape of chest, 26, 30, 32 : movements of chest, 38 : displacement of heart, 42 : vocal thrill, 47 : uncovers heart, 86 : vascular murmurs, 178 : puncture of cavity, 181 : pneumothorax, 209 : diagnosis from pleurisy with effusion, 225 : simulated by collapse of lung, 199, 200 : by pleurisy, 205 : by loculated pneumothorax, 210 : by cancer of lung, 249 : by dilated bronchi, 255 : by actinomycosis, 254 : by cirrhosis of lung, 257. *See* Destructive pneumonia

Physical examination, 1 : method of, 3 : signs, 1

Pigeon breast, 16 : followed by emphysema, 24

PIORRY, theory of percussion, 57 : method, 59 : osteal tone, 63 : humoral sound, 74 : percussion resistence, 74 : hydatid thrill, 75 : ægophony, 100

Pitch of tones, 62

Pitch-pipe, use of, 101

PITRES, signe de sou, 218

Plessigraph, 60

Pleural adhesion, 232 : shape of chest, 30, 32 : movement of chest, 38 : heart uncovered, 86 : mediastinum displaced, 176

Pleural effusion, 214 : shape of chest, 27, 31 : movement of chest, 38 : displacement of heart, 42 : vocal thrill, 46 : relaxation of lung, 82, 84 : percussion dulness, 84 : depression of diaphragm, 175 : displacement of mediastinum, 176 : puncture of chest, 179 : bronchial breathing, 120 : diagnosis from collapse, 200, 201 : from pneumonia, 237 : from hydatids, 253 : from cancer, 250 : from actinomycosis, 254 : from pericardial effusion, 269 : in contracting cancer of lung, 249. *See* Empyema, Hydrothorax, Pleurisy, Pneumothorax

Pleural friction. *See* Friction

Pleural sounds, 125

Pleurisy, 204 : with effusion, 214 : movements of chest, 38 : rale, 187, 205 : simulates phthisis, 205 : in embolic pneumonia, 241 : diagnosis from cancer, 250. *See* Empyema, Pleural effusion, Pleural adhesion

Pleximeter, 59

Plugging of trachea and bronchi, 202 : diagnosis from catarrh, 187 : shape of chest, 31 : movements of chest, 37

Pneumatometer, 35

Pneumoconiosis, 248

Pneumonia, 234 : lobar, 234 : lobular, 238 : destructive, 239 : gangrenous, 239 : embolic, 241 : crepitant rale, 122 : after plugged bronchi, 203 : diagnosis from pleural effusion, 225, 237 : simulated by acute tuberculosis, 243 : by œdema, 190. *See* Destructive pneumonia

Pneumopericardium, 86, 129, 271

Pneumothorax, 206 : shape of chest, 28 : movements of chest, 38 : percussion tone, 68, 82 : amphoric sounds, 128 : false murmurs in, 157 : simulated by catarrh, 187 : by phthisis, 246 : diagnosis from emphysema, 194 : loculated, 210 : in embolic pneumonia, 241 : in pulsating empyema, 230 : in hydatid, 253

POLAILLON, congenital defect of diaphragm, 211

POPHAM, præsystolic jugular pulsation, 165

Position of body, effect on murmurs, 147

POWELL, displacement of mediastinum, 177

Præsystolic impulse, 50 : murmurs, 144, 146, 276, 286 : pulsation in jugulars, 165 : thrills, 53 : venous murmurs, 171

Pregnancy, heart murmurs, 292

Pressure, effect on murmurs, 147

Pterygoid. *See* Alar
Puerile breathing, 112, 117
Puff in breathing, 114, 118. *See* Veiled puff
Pulmometer, 35
Pulmonal percussion tones, 63
Pulmonary. *See* Lungs
Pulmonary apoplexy. *See* Hæmorrhage
Pulmonary artery, position of orifice, 142 : embolism of, 241 : rupture of aortic aneurysm into, 302. *See* Aneurysm
Pulmonary obstruction, 289 : thrill, 52
Pulmonary percussion sound, 77, 82
Pulmonary region, percussion of, 77, 81
Pulmonary regurgitation, 287 : thrills, 52
Pulsatile crepitation, 158 : pleural friction, 158 : respiratory sound, 156
Pulsating cancer, 251
Pulsating empyema, 229
Pulsating pneumonia, 236
Pulsation, aneurysmal, 297 : epigastric, 171 : jugular veins, 164
Pulse-breath, 157
Puncture of chest, 179 : in pleural effusion, 224 : in hydatid disease, 181, 253
Pyæmic infarctus of lung, 241
Pyopneumothorax, 206, 210

Rales, 121 : amphoric, 129 : pleuritic, 187, 205, 214 : pulsatile, 158
RANSOME, stethometry, 35
Recession, epigastric, 173 : of chest in heart region, 40, 43. *See* Inspiratory dyspnœa
Reduplication, heart sounds, 149 : murmurs, 150
Regurgitant murmurs, 140
Relaxation of lung, 71

Resistence in percussion, 74, 85. *See* Percussion in Table of Contents
Resonance in percussion, 68. *See* Tone, Vocal resonance
Resonators, 60
Respiration, movements, 34: wholly abdominal, 38: wholly thoracic, 38: sounds, 111: effect on heart murmurs, 148: pulsatile sounds, 156
REYNAUD, pleural effusion, 46: pleural friction, 126
Rhinophony, 108
Rhonchus, 121, 124
RICHARDSON, auscultation of œsophagus, 183
Rickets, shape of chest, 19: causes pigeon breast, 16: constricted chest, 15: pulmonary catarrh, 186
RINGER, signs in veins of neck, 171

SAVART, fluid veins, 94
SCHREIBER, venous murmurs, 171
Scoliosis, shape of chest, 18, 31
Second sound, accentuated pulmonary, 136, 191
SENAC, pericardial effusion, 268: pericardial fluctuation, 54: pulsation of liver, 172
Septum ventriculorum, perforate, 141, 146
SHAFTESBURY, quoted, 102
Shoulder-blade friction, 133
Shoulder-joint friction, 132
Shrinking, local of chest, 32
Sibilant rale, sibilus, 124
SIBSON, peritonæal friction, 132: aneurysm of Valsalvian sinus, 297
Signe de sou, 218
SKERRITT, conduction of sounds, 183
SKODA, theory of percussion, 57: full sounds, 62: tympanitic sounds, 62: veiled puff, 115, 118
Solidification of lung, 105, 109, 120
Sonorous rale, 124

Souffle. *See* Puff
Sound, in general, 61. *See* Heart, Percussion, Respiration
Spirometer, 35
Splashing sound, pleural, 130, 131: pericardial, 155
Spleen, percussion dulness, 79: in pleural effusion, 218
Sputa, stinking, 240
Stethograph, 35
Stethometer, 35
Stethoscope, 91
STOKES, pulsating cancer of lung, 251
STOLL, percussion, 56
Stomach, percussion, 65, 79
STONE, ægophony, 107
Subclavian murmurs, 161, 248
Subcrepitant rale, 123
Subtympanitic percussion sound, 63
Succussion splash, palpable, 48. *See* Splashing sound
Superficial percussion, 75
SWIFT, quoted, 198
Syphilis of lung, 248
Systolic impulses, 39, 41, 48: heart murmurs, 144, 146, 273, 281, 284, 285, 289: recession of chest, 40, 43: thrills, 52: venous murmurs, 171: jugular pulsation, 166: arterial murmurs, 161

Thoracometer, 35
Thrill, aneurysmal, 299, 302: percussion, 75: valvular, 51: vocal, 45
Thymus, enlarged, 295
Tinkle. *See* Metallic tinkle
Tone, 61, 64, 66
Tonsils, enlarged, cause pigeon breast, 17
Trachea, compression by aneurysm, 301. *See* Plugging
Tracheal percussion tones, 63, 82

Tracheal tugging, 300
Transverse constriction of chest, 14
TRAUBE, on aortic obstruction, 284
Tricuspid obstruction, 286: thrill, 53
Tricuspid regurgitation, 285: venous pulse, 166, 172
Tricuspid valve, position, 142
TROUSSEAU, bell sound, 130
Tuberculosis of lungs, 242: simulated by empyema, 229. *See* Phthisis
Tubular breathing, 114, 118: percussion tones *See* Tracheal
Tumours, abdominal, movements of chest, 38. *See* Aneurysm, Cancer, Hydatids, Mediastinal
Tuning fork, 60
Tympanitic tone, 62, 82
TYNDALL, sound, 61

Valsalvian sinus, aneurysm. *See* Aneurysm
Valves of heart, 142
Valvular murmurs, 140: thrills, 51
VAN SWIETEN, alar chest, 13: crepitant rale, 88, 124
Veiled puff, 115, 118
Veins. *See* Femoral, Jugular, Pulsation, Venous
Vena cava, rupture of aneurysm into, 168, 302
Venous hum, 170: pulsation, 164
Ventricle, rupture of aneurysm into, 302
Vesicular breathing, 111, 117
VIEUSSENS, pulse of aortic regurgitation, 282
Vocal resonance, 96: thrill, 45
Voice, 101

WADE, anastomotic aortic aneurysm, 152
WALSHE, cracked-pot sound, 73: emphysematous friction, 127: pectoriloquy, 105
WARD, venous hum, 170

Water-wheel sound, 157, 271
Watson, hæmothorax, 226
West, S. paracentesis of pericardium, 181
Whispered bronchophony, 99
Whooping cough. *See* Hooping cough
Williams, crepitant rale, 121: tracheal tone, 63
Willis, Thos., metallic tinkle, 88, 129
Wintrich, tuning fork, 60: Skoda's terminology, 63: cracked-pot sound, 73: ægophony, 107: variable pitch of cavernous percussion tones, 245
Woillez, cyrtometer, 8

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